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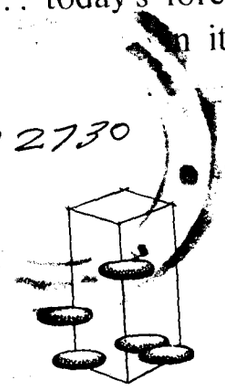

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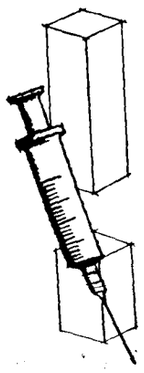

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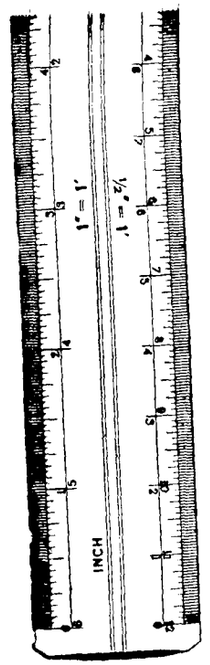

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THE INDIAN JOURNAL OF SURGERY

Vol. XX JUNE 1958 No. 3

EXTRA-PERITONEAL APPROACH FOR PORTO-CAVAL ANASTOMOSIS FOR PORTAL HYPERTENSION*

BY

** V. K. SAINI AND R. H. BETTS, VELLORE (S. INDIA)

It had been suspected for a long time that portal venous pressure might be increased in patients who have splenomegaly and bleeding from oesophageal varices. It remained for Whipple to measure this pressure directly and establish this point in 1936. Out of such early works of Whipple[1], Blackmore[2] Linton[3] and Child[5] etc., have grown the surgical procedures for the establishment of shunts between the portal and caval venous systems. The chances of a portal hypertensive patient having cirrhosis of Liver being alive one year following the first haemorrhagic episode has been variously estimated between 20 percent (Snell)[6] and 50 percent (Patek)[7]. Recently Palmer[8] has reported that 50% of their control cases bled those that had no bleeding before they were recommended for surgery. 70 percent of patients who gave history of haematemesis before being recommended for surgery bled subsequently. In contrast less than 15 percent patients bled after surgery who had bled before and none bled who never had haematemesis before surgery. There is similar convincing proof in the literature that portal decompression reduces the chances of future variceal Haemorrhage and the cirrhotic patient with portal hypertension can be expected to live longer and fare better with a portal shunt than without. It offers an important protection at a reasonable risk of operative mortality and it creates no important post-operative burden. Most of the patients can return to active life. Of course, it does not cure the disease.

Whipple in 1941 proposed that patients who have portal hypertension be classified into two groups, according to the site of obstruction, namely :

(1) *Intra hepatic* : due to various types of cirrhosis of liver—Case 1.

(2) *Extra hepatic* : due to changes usually in the portal vein itself, some congenital, some as a result of trauma or infection and some of undetermined cause—Case 2.

In addition Hellenbeck [9] states that occasional patients are encountered with enlarged spleen and bleeding from oesophageal varices who have no clinical or laboratory evidence of hepatic disease, who have portal hypertension with no evidence of extra-hepatic portal obstruction (our case 3). In these patients liver feels more firm and contains more fibrous tissue than is normal, although it does not have the regenerative nodules and other criteria necessary for a diagnosis of cirrhosis. These patients probably represent unusual type of intrahepatic portal obstruction.

Table I

Cause of Portal Hypertension	Number of patients	Male	Female	Age at Operation
Intra Hepatic—				
(*a*) Cirrhosis	5	5	...	18—45
(*b*) Unknown (Hellenbeck)	2	?	...	7—18
Extra-Hepatic —				
(*a*) Cavernous transformation Congenital	3	3	...	6—15
(*b*) Thrombosis	...	...	...	...

Table I shows the types of cases seen in our small series.

* Read at the Sectional meeting of the Thoracic Surgery Section of the Association of Surgeons of India held at New Delhi, on 15th and 16th November, 1957.

** From the Department of Thoracic Surgery, Christian Medical College Hospital, Vellore, S. India.

According to the present concept, whatever may be the cause of portal hypertension, the increased portal pressure may lead to congestive splenomegaly and oesophageal varices, which may bleed.

Shunts between the portal and caval systems are made with the objective of providing a direct outlet for portal blood, that will offer sufficiently less resistance to flow of portal blood, than do the collateral channels provided by nature. The shunt then minimises the use of these collateral channels and thus serious bleeding from the varices is prevented.

To obtain this result, it has to be presupposed that the shunt will be large enough to accomplish its purpose. It is very difficult to state accurately the minimal size of an effective shunt, but it can be said that minor shunts between small branches of the two venous systems are unsatisfactory.

The subject of condemning splenectomy for portal hypertension unless at the time surgeon is prepared to establish a splenorenal shunt, cannot be more emphasized than the impressive remarks of Dr. Allen O. Whipple himself.

> " For many years one of our most discouraging follow up problems in our spleen clinic at the Presbyterian Medical Centre has been that of dealing with recurring haemorrhage in patients who had had splenectomy for congestive splenomegaly or Banti's syndrome."[10]

So, in short, any surgical interference for portal hypertension should not be other than a shunt operation. Splenectomy may only be indicated in rare cases of congenital cavernous transformation of portal vein. The two types of shunt that are being done these days are :

I. Spleno-Renal.

II. Porto-Caval.

Table 2 shows the types of shunts done at the Christian Medical College Hospital.

Table 2

Type of Shunt	Patients	Operations done
Spleno-renal	8	8
Portocaval	1*	1
Splenectomy ...	2	3**

*Patient had previous Spleno-renal Shunt.

**Patient had oesophageal Ligation of varices.

At present it is impossible to set any hard and fast rule by which to determine which type of shunt should be performed. The logical approach at present seems to be that a surgeon undertaking the treatment of portal hypertension must be prepared to perform whichever type of shunt is indicated by the situation arising from the type of a particular case. Palmer and others believe that porto-caval shunt is considerably more effective than spleno-renal in preventing further variceal haemorrhage. This is evidenced by their statistics, that 7 patients out of 12 had subsequent bleeding episodes after a Spleno-Renal shunt while only one patient out of 45 bled who had Porto-Caval. The proponents of Spleno-Renal shunt argue, because of the low operative mortality and easy technical procedure. Also it allows the removal of large hypersplenic spleen and thus hypersplenism if it be present is promptly relieved, and in addition a considerable amount of blood is prevented from entering the portal system. On the other hand, proponents of Porto-caval shunts state that two factors might favour having the spleen in place : this organ shrinks in size following adequate portocaval shunt, and the large collaterals between the spleen and lateral body wall are preserved. Here in India quite a few patients with portal hypertension have big spleens. We have been removing these large spleens and thus prefer to do a Spleno-renal in these cases. As yet we have not done a Porto-caval shunt in a patient with a large spleen, so as to tell as to what happens to the large spleen.

In general, however, we agree that portocaval shunt is the most useful procedure in patients with cirrhosis. They usually have small spleens but when the block is outside the liver, the spleno-renal shunt is preferred, for the portal vein is frequently compromised by a thrombus, and therefore useless for purposes of portal decompression.

The selection of patients for operation is done according to the criteria that were aptly proposed by Linton[1] a few years ago.

The present extra-peritoneal approach for portocaval anastomosis, that is being presented is developed because of the tedious dissection of porta-hepatis in a large number of cases. These patients who have ascites, usually have extensive adhesions between the bowel especially around porta-hepatis, and freeing of these vascular adhesions is a very haemorrhagic process. Moreover the collateral vessels

that develop along the portal vein are usually anterior to this vessel and so dissection in the free margin of lesser omentum can be a bloody procedure. The idea of this approach was derived from this section of abdomen (Fig. No. 1) at the level of the portal vein in

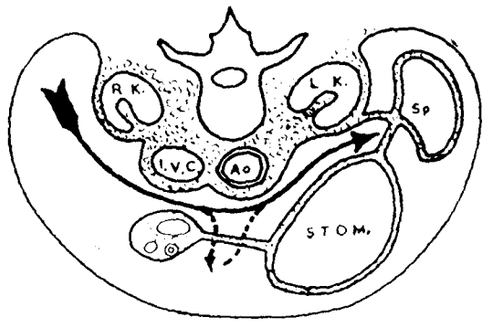

Fig. 1

the lesser omentum which shows the close proximity of these two vessels. The portal vein also lies posteriorly in the lesser omentum and so a posterior approach was justified to be attempted. This procedure gets away from the hazard of tedious intra-peritoneal dissection and the surgeon does not have to fight with the intestines.

This paper is being presented to describe this new approach and also report three interesting cases of portal hypertension.

So far as we have done nine shunt operations at the Christian Medical College Hospital, 1950–57.

Splenorenal ... 8

Porto-Caval ... 1

Our operative mortality has been nil.

Technique

Hypothermia : We have started operating on cirrhotic patients under hypothermia of moderate degree, for we believe that the damaged cirrhotic liver stands the insult of anaesthesia and surgery better under this cooling. We place the patient on the cooling blanket after induction of anaesthesia and intubation. The patient is positioned and we start operating. The patients gradually cool to 34 °C. when the cooling is turned off and then temperature allowed to drift a few degrees. Thus we obtain around 32 °C. temperature which is a very safe range of hypothermia.

A right thoraco-abdominal incision gives the best exposure. Patient is placed in the left semi-lateral position, and a long incision is made over the 8th intercostal space, crossing the costal margin and terminating near the umbilicus. The right rectus muscle and the abdominal muscles lateral to it are transected upto the peritoneum. The chest cavity is entered through the eighth intercostal space. Adequate exposure is obtained through the interspace and so no rib is excised. The costal margin is cut across. The wound is then partially held open by a rib spreader. The diaphragm is then mobilised from the liver. The diaphragm is then divided in the line of the cutaneous incision, to a point near the central tendon. The rib spreader is now opened more and a good exposure is obtained.

The liver is now lifted anteriorly and at the same time delivered a little into the chest. Extra-peritoneal dissection is started bluntly in the plane anterior to the kidney. The peritoneum is thus separated from the postero-lateral abdominal wall and is carried down over anterior to the kidney until the inferior vena cava is exposed. The peritoneum thus reflected carries with it the hepatic flexure of the colon and duodenum. This part of the exposure is carried out without difficulty and a good exposure of the I.V.C. with its right renal branch is obtained. The vena cava is then followed upwards, clearing it upwards up to the liver.

The exposure and identification of portal vein is slightly difficult, but it should be remembered that portal vein crosses the vena cava obliquely anterior to it before it reaches the porta-hepatis (Fig. No. 2). So at this time if

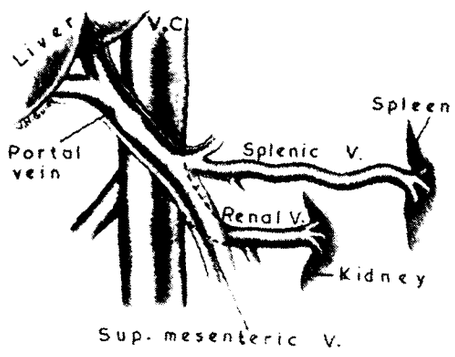

Fig. 2

the angle between the liver and vena cava is dissected, entering the lesser sac in this region portal vein is identified, lying posterior to the common bile-duct. Usually the Hartmann's pouch of the gall bladder is identifiable without much difficulty. If this be followed to the common bile-duct—the portal vein can be located posterior to it. The portal vein is then dissected both towards the portal-hilum, and distally. The two divisions of the portal vein are isolated where they enter the liver. A heavy silk ligature is placed on each of the divisions. A Blalock clamp is then placed on the portal vein distally and the portal vein divided just before it divides into its two branches to the liver. All adventitious tissues are separated from the portal vein and an adequate length is obtained to bring it down to the vena cava. Direct portal pressures are recorded in the portal vein before it is divided.

A portion of the inferior vena cava is then isolated by a Satinsky clamp. An incision is made on this vena cava and an end-to-side anastomosis is carried out with the portal vein. A continuous over and over 00000 silk suture is used posteriorly and this is continued as a continuous everting mattress (or interrupted everting mattress sutures may be used in children) anteriorly. The portal pressures are again recorded which will show considerable fall.

The closure is then in the usual fashion, closing the chest and abdomen. The chest is drained by an intercostal drainage tube for 24–48 hours.

CASE REPORTS

Case No. 1 : Nageswaran, I.P. No. 46987

35 years old male admitted to the medical ward on 12–6–57 with Haematemesis of 5 days duration. On admission patient was having Malaena. He gave a history of a previous episode of Haematemesis 5 months ago for which he was hospitalised in Coimbatore for 3 months. He also had occasional Malaena for past 5 months. Patient was admitted in profound anaemia, HG. of 2.0 gms. He had an enlarged spleen palpable 3 F.B. below the left costal margin. No ascites. Patient was treated by repeated blood transfusions and by 18–6–57 his HG. was 7 gms. Patient still continued to have Malaena.

20–6–57 Ba. Swallow showed oesophageal Varices—Fig. No. 3.

21–6–57 A Spleno-portogram was done. Splenic Pressure 28.5 cms of water– Fig. No. 4.

Spleno-portogram shows dilatation of splenic and portal veins. There is poor visualisation of intra-hepatic vessels. Large number of collateral vessels can be seen.

A diagnosis of cirrhosis of liver with bleeding oesophageal varices was made and patient was referred to us and transferred to thoracic service with a HG. of 11.25 gms. When still waiting for surgery patient had more Malaena and his HG. dropped again to 8.5 gms. He was prepared with further blood transfusions. His liver function studies were :

Ictric Index	: 5 Units.
Total Protein	: 4.65 gms.
Albumin	: 2.45 gms.
Globulin	: 2.20

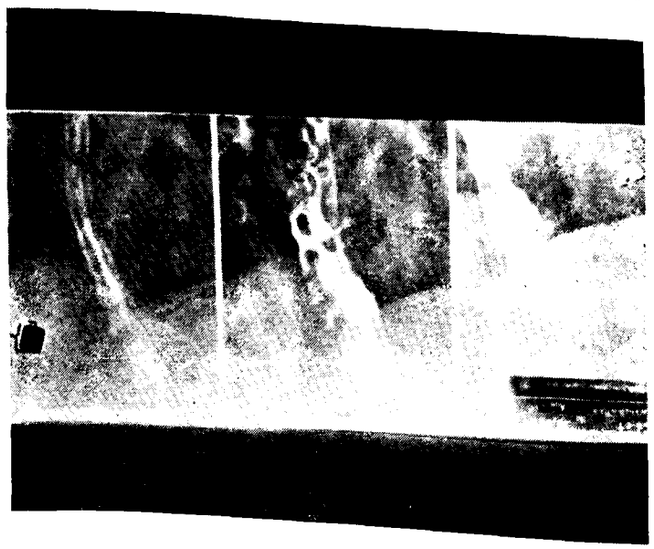

Fig. 3
Barium Swallow of Case No. 1 showing oesophageal varices.

Fig. 4
Spleno-portogram of Case No. 1 showing dilated splenic and portal veins.

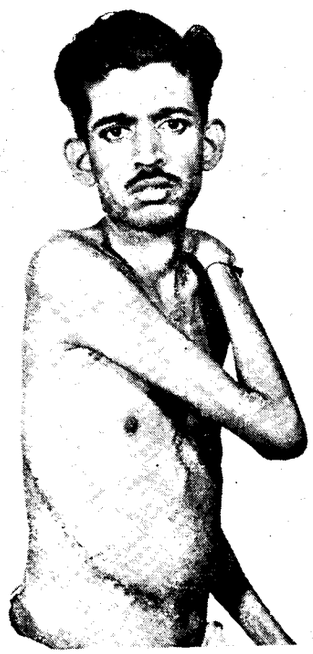

Fig. 5
Photograph of Case No. 1, shows the incision used for extra-peritoneal porto-caval anastomosis.

Prothrombin Time	: 15 : 15
Alkaline Phosphatase	: 6 Units.
Thymol Turbidity	: 1 Unit.
Thymol Flocculation	: 0
Zinc Turbidity	: 1 Unit.

On 24–7–57 a Splenectomy and a Spleno-renal anastomosis was done by Dr. Gopinath through a left thoraco-abdominal incision. Portal pressures were 28 cms of saline, liver showed. Hobnail appearance.

Surprisingly there was no drop in portal pressure following the anastomosis. We are not able to explain why this happened.

Post-operatively patient again had bouts of haemetemesis and Malaena. HG still ranged at 7 gms and repeated blood transfusions were required.

So on 31–8–57 we did a Portocaval Anastomosis through the right extra-peritoneal approach under mild hypothermia. Pressure in the portal vein registered 29 cms. of saline which dropped to 16 cms of saline after anastomosis. Following the second operation patient had an uneventful recovery and patient was discharged on 18–9–57. No further bleeding so far.

Liver Biopsy : Negative but grossly liver had hobnail appearance.

Spleen : Splenectomy with siderotic nodules.

Comment : A spleno-renal shunt in this patient failed to decompress the portal hypertension. So a porto-caval anastomosis was done which successfully decompressed it and no further bleeding occurred.

Case No. 2 : P. Bhatan—I.P. No. 50167

15 years old boy admitted to medical ward at the Christian Medical College Hospital on 4–9–57 with history of Haematemesis for 2 days (July 14th and 15th) and had tarry stools for many days. He had fever for one day prior to bleeding. He was admitted to the local hospital and was given Plasma. He gave history of previous episode of Haematemesis, when 5 years of age. He had fever for one week prior to this, bleeding episode. Remaining past history was negative.

G.P.E. Showed a well nourished boy. No Jaundice. Spleen palpable 3 F.B. below L.C.M. There was a systolic murmur in all areas. Rest of physical examination was negative, except for scabies infection.

LAB Data :

HG	: 12.5 gms.
WBC	: 12.500
P	: 48
E	: 20
L	: 32
Prothrombin Time	: 16 seconds

Control	:	15 seconds
Ictric Index	:	4
Total proteins	:	7.1
Alb.	:	3.35
Glob.	:	3.75
Alkaline Phosphatase	:	14 Units
Thymol turbidity	:	2 Units.
Thymol Flocculation	:	0
Zinc Sulphate turbidity	:	5 Units.
Bromo sulphaline retention 3%	:	1st hour.
Barium Swallow	:	Shows filling defects due to oesophageal varicosities.

A diagnosis of Portal hypertension was made, probably extrahepatic and a Spleno-Portogram was done on 12-9-57 which shows complete absence of the splenic vein shadow. A large number of anastomotic veins are seen in the splenic hilum and some passing upwards into the region of oesophagus—Fig. No. 6.

Splenic pressure—320 mm. of saline.

Radiologist's opinion was of Splenic Thrombosis.

Liver Biopsy : Negative.

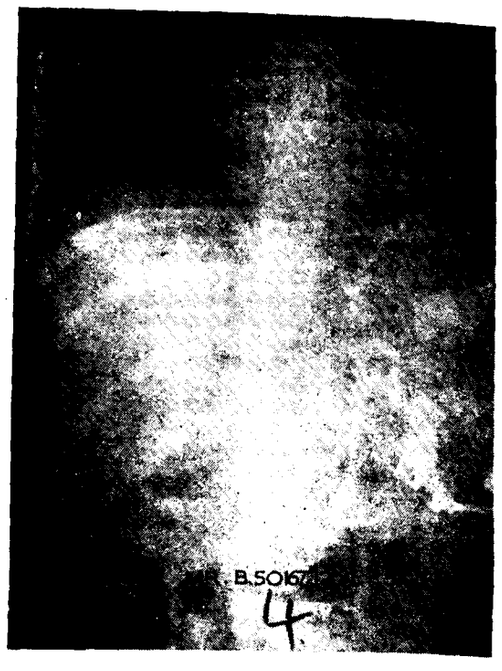

Fig. 6
Spleno-portogram of Case No. 2 shows complete absence of splenic vein.

On 16-9-57 patient was referred to the Thoracic Surgery Department. Our opinion was that in view of haematemesis at the age of five this may be a case of cavernous transformation of the portal vein.

Patient was transferred to us, and it was decided to do a left thoraco-abdominal exploration of the splenic hilum, and if there was thrombosis of the splenic vein—splenectomy be done and also at the same time do a portogram, at the table.

On 26-9-57 patient was explored through the left thoraco-abdominal incision, as for spleno-renal shunt. The spleen was only slightly enlarged. Liver looked normal. Dissection of the hilum of the spleen did not show any thrombosis but instead there were numerous small splenic veins. Portal Pressure registered 38 cms. of saline. Splenectomy was done. Following this a Portogram was done on the table by passing a polyethylene tube through a mesenteric vein of a loop of Jejunum—Fig. No. 7.

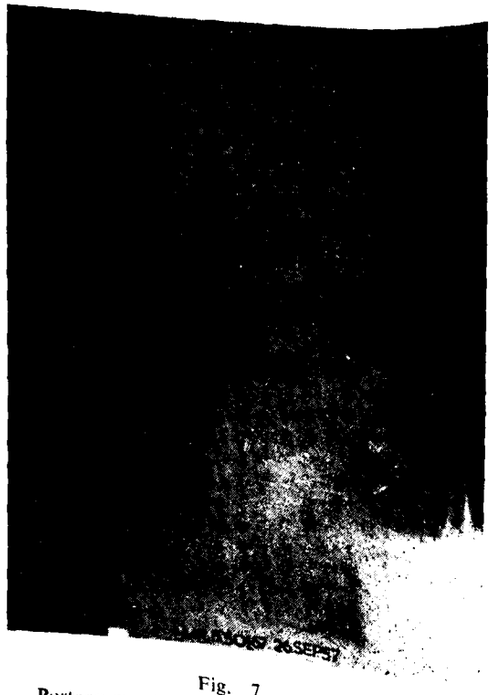

Fig. 7
Portogram of Case No. 2 shows Cavernous transformation of portal vein.

This shows cavernous transformation of portal vein. The portal pressure now had dropped to 28 cms. of saline.

Liver Biopsy : was negative and spleen showed fibrosis.

Comment : A case of congenital cavernous transformation of the portal vein is presented.

Case No. 3 : Prabakaran - I.P. No. 50382

7 years old boy was admitted to the hospital on 11-9-57 with the complaint of abdominal swelling of 4 years duration. Parents first noticed this swelling in 1953 on the left side. This has gradually been increasing in size, and for the last 3-4 months the swelling has increased rapidly.

In 1954 patient had high fever. Haematemesis and tarry stools for 2 days. He was hospitalised in Salem.

In August 1956, patient had another episode of Haematemesis for 3 days with high fever. He was again hospitalised for two weeks. Past history and family history was negative.

Physical examination showed a fairly nourished, anaemic boy. Positive finding being a large spleen extending upto the umbalicus. No ascites, liver not palpable.

Laboratory investigations at admission were :

Haemoglobin	:	5.25 gms.
P.C.V.	:	25%
WBC	:	2,600
Polys	:	40
Eosinos	:	9
Lymphs	:	51
Platelets	:	104,000
Coagulation Time	:	5 mnts. 38 seconds.
Bleeding Time	:	2 mnts. 35 seconds.
Prothrombin Time	:	16 second (Control 14 seconds)
Sedimentation Rate	:	1st hour 13. Second hour 32.
Urine and Stools	:	Negative.
Liver Function tests :		
Icterus Index	:	3 Units.
Total Proteins	:	7.4 gms.
Alkaline Phosphatase	:	8.4
Zinc Turbidity	:	10 Units.
Thymol Turbidity	:	3 Units.
Thymol Flocculation	:	+
Barium Swallow	:	Did not show any varices.

The Paediatrician thought that in view of the pancytopaenia this case was that of Hyper-splenism.

On 26-9-57 a Spleno-portogram was done. Splenic Pressure was 40 cms. of water. The splenogram does not show any portal obstruction, but we believe that portal radicals are rather large than normal. A few intercostal veins are seen, which means that blood is flowing through the collateral channels— Fig. No. 8.

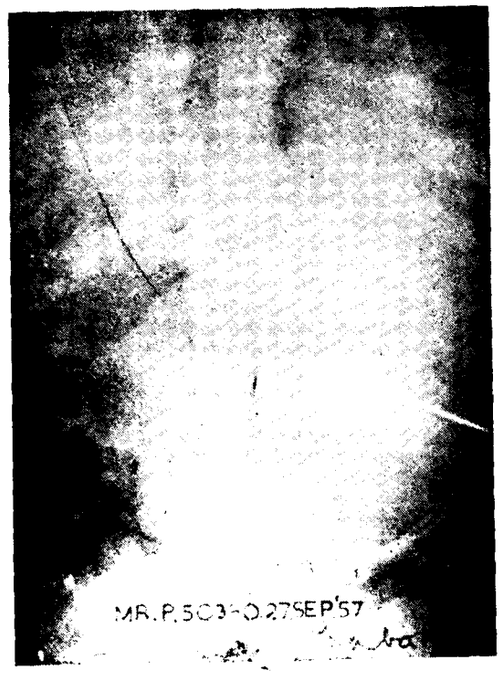

Fig. 8
Spleno-portogram of Case No. 3.

On 28-9-57, patient vomited large quantity of blood and had tarry stools. He was transfused with blood. He was also referred to us for opinion.

Our opinion was that this patient had portal hypertension and so should have portal decompression as soon as his general condition would allow. We believed that he should have his large hypersplenic spleen removed and at the same time have a spleno-renal shunt.

Patient was prepared by blood transfusions and on 5-10-57, Splenectomy and a Spleno-renal shunt was done through a left thoraco-abdominal incision. Liver felt absolutely normal. Spleen was greatly enlarged. Portal Pressure registered 42 cms. of saline before and 23 cms. after decompression. Patient had an uneventful recovery and was discharged on 25-10-57.

Liver Biopsy : Showed increased fibrosis, but no cirrhosis.

Comment : Here is a case of Portal Hypertension, who has no cirrhosis or extra-hepatic block. We believe that probably this case falls into Hellenbeck's intrahepatic portal hypertension of unknown etiology.

Table 3

S.No.	Cases	Haematemesis	Ascites	Oesoph Varices	Liver Biopsy Cirrhosis	Spleen	Portal Pressure before surgery	Portal Pressure after surgery	Haematemesis after surgery	Type of Shunt	Date	Remar
1.	Webber—96377 45 years.	Yes	Yes	Yes	Yes	Neg	...	...	Yes	Spleno-renal.	8 3 50	Infec. Hepatitis rhosis. Died
2.	Dora Raj—18016 23 years	Yes	Yes	Yes	Yes	*	420	400	Nil	Spleno-renal.	Anastomo-sis 15 11 54 Autopsy	2-1-51 Thrombosed o
3.	Marudavanam—32551 18 years.	Yes	Yes	Yes	No biopsy	Very big	...	...	Nil	Spleno-renal.	8 6 56	Cirrhosis.
4.	Prasad Singh—36023 22 years.	Yes	Yes	Yes	Pos	7 F.B.	320	300	Nil	Spleno-renal.	26 9 56	Cirrhosis
5.	Kumar—36277 6 years.	Yes	Nil	Yes	Neg	3 F.B.	460	480	Yes	Splenec-tomy.	24 10 56	Cavernous tran mation. Lig oesophageal ces—31-10-5
6.	Radhakrishnan— 14 years.	Yes	Nil	Yes	Neg	4 F.B.	360	275	Nil	Spleno-renal.	12 3 57	Cavernous formation.
7.	Pandurangan—47341 18 years.	Nil	Nil	Nil	**	1500 gms.	340	240	Nil	Spleno-renal.	30 7 57	Hallenbeck's hepatic obs tion.
8.	Nageswaran—46987 35 years.	Yes	Nil	Yes	Neg	3 F.B.	280	280	Yes	Spleno-renal.	24 7 57	Grossly liver sh
9.	Bhatan—50167 15 years.	Yes	Nil	Yes	Neg		290	160		Porto-caval.	31 8 57	Hobnail but bipsies showe cirrhosis.
10.	Prabhakaran—50382 7 years.	Yes	Nil	Yes	Neg	3 F.B. Very big	380	280	Nil	Splenec-tomy.	26 9 57	Cavernous tran mation.
							420	230	Nil	Spleno-renal.	6 10 57	Hellenbeck's hepatic obs tion.

*Slightly enlarged.

**Chronic Hepatitis

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SURGERY OF INTRACRANIAL VASCULAR LESIONS

BY

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Surgery of intracranial vascular lesions is a fascinating subject. Enormous progress has been made in the treatment of these conditions during the past two decades especially after the advent of cerebral angiography. Before cerebral angiography, diagnosis was difficult and these lesions were thought to be rare. Since angiography has become a routine procedure, these lesions are diagnosed more easily. Our knowledge has advanced with better understanding of the origin and pathology of these conditions. Treatment has progressed by better technique and by better methods of anaesthesia. This paper deals with the experience of the Neurosurgical Unit, General Hospital, Madras from the year 1951 to 1957. The following topics are considered under intracranial vascular lesions.

(1) Arterio-venous malformation
(2) Aneurysms
(3) Vascular tumours
(4) Carotid Thrombosis
(5) Other conditions like Sturge Weber Syndrome.

Out of a total of 850 cases of cerebral vascular diseases admitted in the Government General Hospital, Madras from 1951 to 1957 the following lesions were found.

Table 1

Serial Number	Type of lesion	Number of cases
1.	Arteriovenous Malformations...	9
2.	Aneurysms	53
3.	Vascular Tumours	41
4.	Carotid artery thrombosis ...	9
5.	Sturge Weber Syndrome	6

1. *Arteriovenous Malformations :* Arteriovenous malformations of the brain have been known hitherto by various names like hamartoma, angiomatous malformation, arteriovenous aneurysm etc.

Telangiectases and cavernous malformations are usually found as incidental autopsy findings. They do not cause signs during life. Rarely, they may cause subarachnoid haemorrhage. These may also rarely occur in the brainstem and may stimulate a brain tumour. Out of a total of 7000 autopsies 10 cases of telangiectasis and venous angiomas of the brain were found.

The chief abnormality that is often met with is the arteriovenous angioma (the one caused by a direct entry of an artery into a vein). This leads to an increase in the venous tension and the veins thicken and enlarge. During the course of years such a malformation increases in size and may assume large proportions. This is due to the pressure effects of the malformation on the brain leading to a slow atrophy. Patterson and McKissock[5] suggested that this may be due to absorption of small blood clots and the surrounding nectrotic brain material, leading to a diminution of the support for the malformation and thus leading to increase in size. Cysts and false aneurysms are also frequently seen in association with these malformations.

Such a malformation also acts as a shunt which directs the blood from going to the brain and allows it to empty directly into the venous system without going through the capillaries of the brain. This interferes with the normal blood supply to the surrounding areas of the brain, which thus suffer from the effects of anoxia and gradually begin to lose their function.

This is best exemplified in the following case. Patient P. N.S. 7146 aged 35 years reported with severe headache and gradually increasing weakness of his left upper and lower limbs. There was a bruit on auscultating his scalp and huge tortuous scalp vessels were seen. Plain X-rays showed the prominent impressions of the meningeal vessels on the skull. (fig. 1).

On angiography it was clearly seen that all the dye that was sent into the internal carotid artery was taken up entirely by the posterior cerebral artery. Though the angiograms were repeated twice the anterior cerebral and the middle cerebral systems did not fill up. This

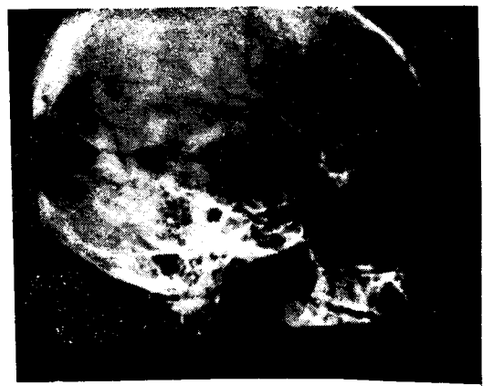

Fig. 1
N.S. 7146—X-rays showing prominent vascular markings in the skull.

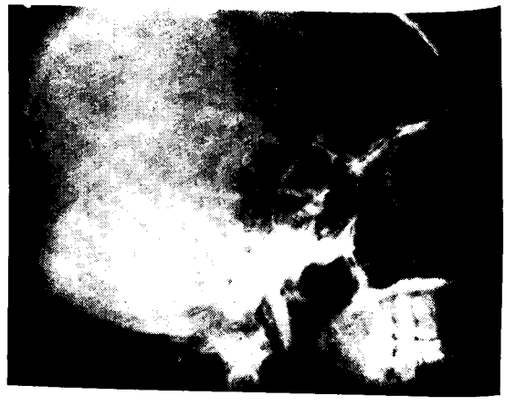

Fig. 2
N. S. 7146– Angiogram– Arterial phase– Blood going through the internal carotid artery drains completely via the posterior cerebral into the arterio venous malformations.

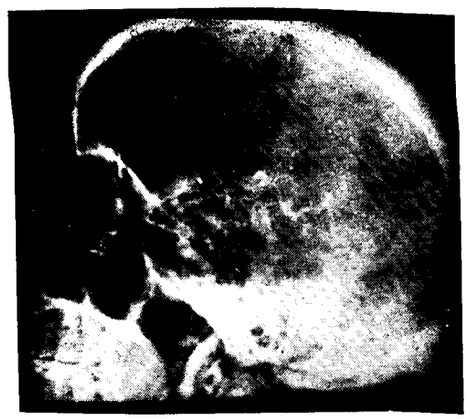

Fig. 3
N.S. 7146—Capillary phase.

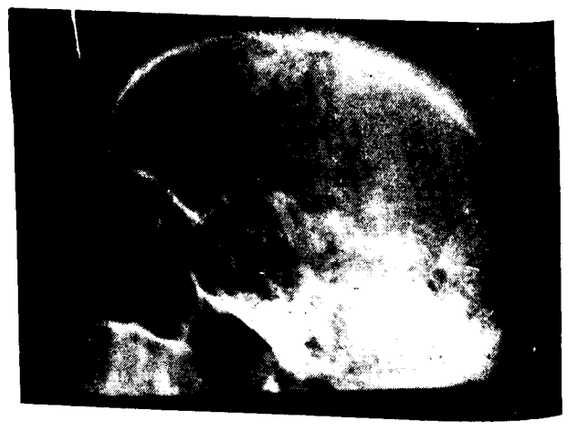

Fig. 4
N.S. 7146– Venos phase –*Note*– that no arteries going to the hemisphere are filling.

clearly demonstrates how huge anterior venous malformations reduce the supply to the adjacent parts of the brain. (Figs. 2, 3 and 4).

Arteriovenous malformations of the brain usually occur in the territory of the anterior cerebral or the middle cerebral arteries. These may be situated in the cortex or in the subcortical region. When situated in the cortex the malformation exercises direct pressure on the cortex leading to cortical atropy. The increased venous pressure interferes with the venous drainage of the surrounding portions of the brain leading to oedema and increased irritability and focal fits.

Occasionally it may so happen that a small vein in the malformation may burst and lead to a subarachnoid or intracerebral haemorrhage. Bleeding from an arterio-venous malformation is usually not so severe as from a ruptured aneurysm. One patient has been reported to have had four non-fatal bleeds.

From the above, the symptomatology of arterio-venous malformation will be clear. The

patient may exhibit a gradually increasing weakness of a limb or the first symptom may be an attack of focal fits. It may also so happen that the paralysis and the weakness may be disproportionately large compared to the size of the malformation. This is due to short circuiting of blood supply that has already been mentioned. Rarely, the first symptom may be a subarachnoid haemorrhage. Periodic migrainous headache, strictly unilateral, may also indicate an arterio-venous malformation. Two out of the nine cases reported here had unilateral headache resembling migraine. Clinical examination may show an audible bruit over the suspected area. If the head is shaved the scalp may show tortuous vessels in some cases.

Table 2

Symptoms of arterio - venous malformations

Serial Number	Symptoms at onset		Number of cases
1.	Focal fits and weakness	...	6
2.	Unilateral Headache...	...	2
3.	Subarachnoid haemorrhage	...	1

Nine cases of arterio-venous malformation have been met with. Six out of these nine cases presented symptoms of focal fits followed by weakness of the limb. Two came with headache and one with subarachnoid haemorrhage. Diagnosis is made by angiography. The focal symptoms may make one suspect a focal space occupying lesion. The absence of papilloedema, in spite of a long history of neurological signs, will exclude any big space occupying mass.

The final diagnosis is possible only by angiography. In an angiogram, the veins are found filled even during the arterial phase. The veins are dilated and tortuous and are bigger than usual. The feeding arteries also are lengthened and tortuous.

The aim of treatment is to release the pressure on the cortex, to bring down the irritability of the surrounding brain and to improve the blood supply. The ideal treatment would be a complete excision of the entire mass which can occasionally be a very formidable procedure. Such a procedure is sometimes impossible, if the removal will lead to a big neurological deficit.

Patient P (N.S. 4798) aged 20 years was admitted to the Neurosurgical Unit with a complaint of fits involving the left upper limb for the past two years. Once in a few days the patient used to get spasms and involuntary movement involving the fingers, forearm and the arm on the left side. Occasionally these spasms used to spread all over and cause a

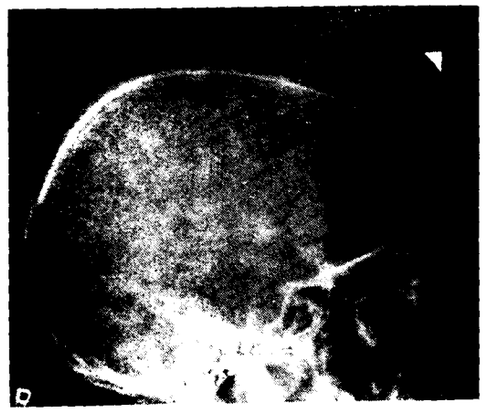

Fig. 5
Prominent vessel marking in the skull.

Fig. 6
Arteriogram showing the arterio venous malformations. See the big veins filling even in the arterial phase.

major attack of convulsions including loss of consciousness.

On examination there was no neurological abnormality except that the jerks in the left upper limb were exaggerated. Plain X-ray showed prominent vessel marking on the skull. (fig. 5). A right cerebral angiogram was done and this showed a big arterio-venousmalformation in the upper portion of the left parietal region (fig. 6–9).

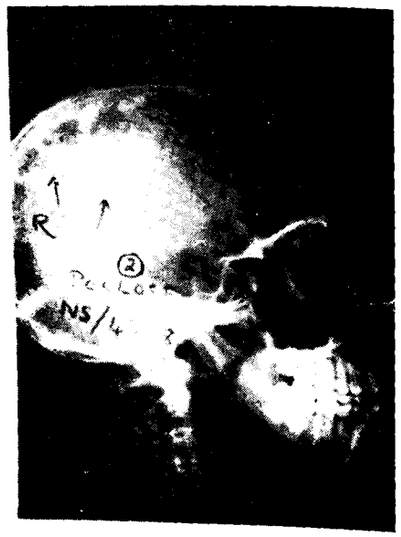

Fig. 7
Venous phase.

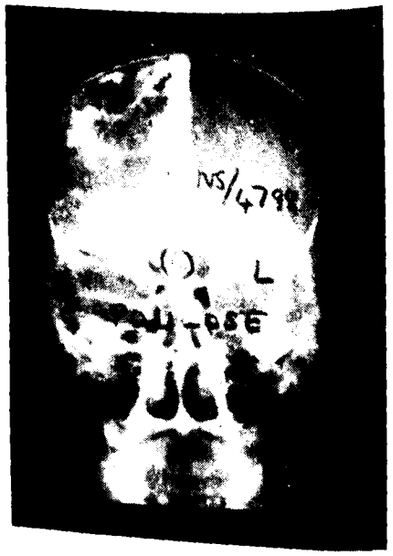

Fig. 9
Venous phase—antero posterior view.

Fig. 8
Arterial phase—antero posterior view.

An osteoplastic craniotomy was done under hypotension anaesthesia and all the main feeding arteries leading to the malformation were clipped. Post-operative angiograms show almost complete disappearance of the mass. (fig. 10–13).

In this case if we had gone on to a complete excision of the malformation, there was great danger of his becoming weak in his limbs. Hence it was decided only to occlude all the major vessels leading to the aneurysm.

The surgeon has to consider all the aspects before deciding on the particular line of treatment. While ligaturing feeding vessels it is necessary to identify the arteries first and clip them. If the draining veins which are as big as arteries are occluded by mistake there may be enormous swelling and the malformation will burst leading to severe haemorrhage and cerebral oedema.

All the 9 cases presented were operated on under hypotension anaesthesia. When hypoten-

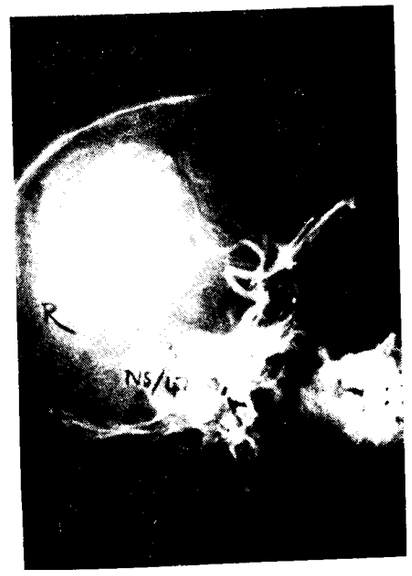

Fig. 10
Post operative angiograms. *Note* that the malformation does not fill and that the size of the feeding artery is much smaller now.

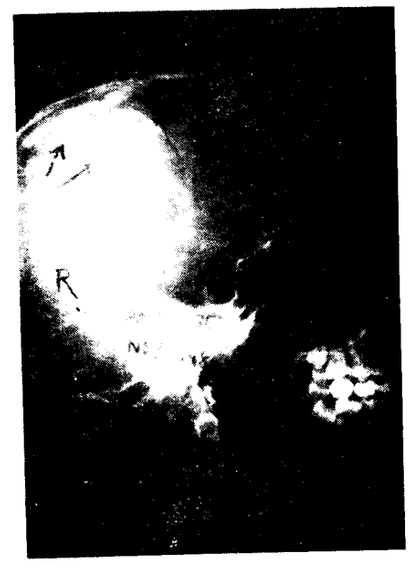

Fig. 11
N.S. 4278 Post operative—venous phase—lateral view.

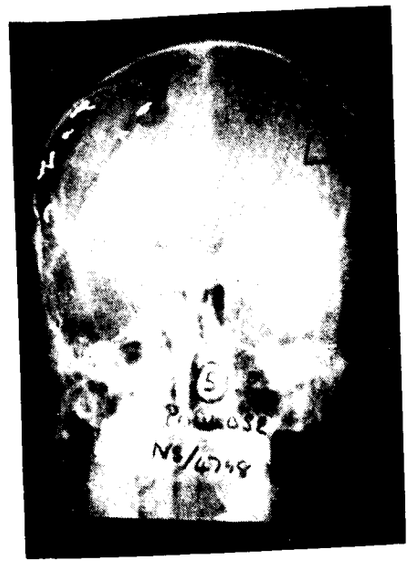

Fig. 12
N.S. 4278 Post operative arterial phase A.P. view.

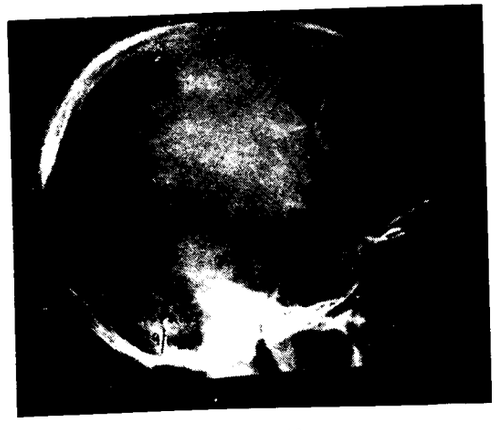

Fig. 13
Patient S.R.P.—Plain X-ray showing calcification inside the skull.

sion is very effective the arterio-venous malformation may collapse and the full extent of the lesion may not be appreciated. In one of the above cases the anaesthetist had to be requested to raise the blood pressure by lowering the head to enable the surgeon to recognise all the leading vessels. Hypotension anaesthesia has been a great boon in the treatment of these cases.

With the advent of hypothermia, we are able to completely excise suitable lesions without great fear of haemorrhage or causing neurological deficits due to cerebral anoxia. In this series only one small arterio-venous malformation in the parietal region was completely excised. In all the rest the leading vessels were clipped with satisfactory results. On follow up, all the cases operated on have done well without any permanent disability.

When arterio-venous malformations occur in the brain stem surgical removal is not possible. One case came with symptoms resembling brain tumour with papilloedema. Angiography showed a small midline arterio-venous malformation. Ventriculo cisternostomy was done and the patient was given deep X-ray therapy. The patient expired one year later.

Value of Deep X-ray therapy : In the treatment of intracranial angiomas and arterio-venous malformations sometimes it becomes necessary to employ deep X-ray therapy. When surgical excision of the angiomas is not feasible or when surgical occlusion of the leading vessels is only partially successful deep X-ray therapy can be usefully employed to diminish the circulation through the malformations. Some consider this procedure to be of no benefit while there are other reports holding that it is beneficial. (Potter)[6].

2. *Intracranial arterial aneurysms :* Patients with intracranial arterial aneurysms may present themselves either as cases of focal neurological disabilities or with subarachnoid haemorrhage. Subarachnoid haemorrhage has been recognised only for the past seventy or eighty years. Before that it was confused with intracerebral haemorrhage commonly known as cerebral apoplexy.

Thomas Willis (1621–1675)[11] and Brunner (1653–1727)[1] noted the importance of aneurysms of small cerebral vessels in the aetiology of apoplexy. Morgagni (1682–1771)[4] considered ruptured aneurysm as an important cause of cerebral haemorrhage. More than 86 papers were published between 1800–1880 but still the association of intracranial aneurysm with subarachnoid haemorrhage was not widely accepted or recognised. Even as late as 1923, Goldflam[3] reviewing 13 cases suggested S.A.H. was due to capillary oozing due to vasculomotor instability. Collier's[2] classical account in 1922 and later extensive reviews by Symonds in 1923[9] and 1924[10] made clear the correct relation of Intracranial aneurysm to subarachnoid haemorrhage.

Haemorrhage into the subarachnoid space is caused by a blood vessel rupturing into the subarachnoid space. This is usually due to a small aneurysm in the blood vessel in the younger age group of patients. In the older age group also it is commonly due to a congenital aneurysm but the factor of arterio sclerosis also comes into the picture. Small aneurysms are due to congenital weakness in the medial muscular wall of the cerebral blood vessel leading to a protrusion of the wall of the blood vessel. This may go on throughout life without endangering the life of the person. In post-mortems a number of intact aneurysms have been found in the circle of Willis, which during life have not caused any symptoms. Syphilitic, arteriosclerotic and mycotic aneurysms are not as common as a congenital berry aneurysm.

Sometimes due to various known and unknown reasons this aneurysm may suddenly rupture. Once it ruptures, the progress of the case depends upon the size of the aneurysm and its site. When a big aneurysm ruptures and there is an enormous quantity of blood thrown into the subarachnoid space it may end fatally.

When an aneurysm ruptures into the ventricle death is inevitable. Sometimes the aneurysms may rupture partly into the subarachnoid space and partly into the brain. When the blood leaks into the brain, it forms an intracerebral haematoma and may cause severe fatal pressure on the brain.

It may so happen that the aneurysms may slowly leak into the cerebral substance and form an intracerebral haematoma. The haematoma may begin to calcify and act like a space occupying lesion.

Mr. S. R. P. was referred to the Neurosurgical Unit with a complaint of inability to see with his right eye. On examination there was optic atrophy in the right eye. There was no other neurological abnormality. X-rays of the skull showed calcified spots in the right suprasellar and frontal regions. (figs. 14 and 15). Cerebral angiogram showed an aneurysm in the

Fig. 14
Patient S.R.P.—Plain X-ray A.P. view.

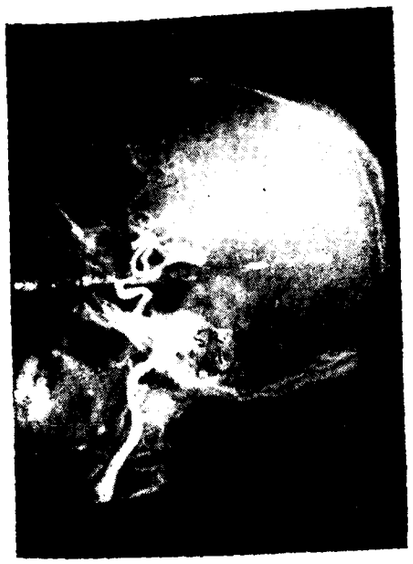

Fig. 15
Patient S.R.P. Carotid angiogram.
Note the aneurysm.

terminal portion of the right internal carotid artery. There was a false aneurysm leading from this sac anteriorly. (fig. 16). The A.P. view showed the lateral extent of the sac. The calcification was inside a haematoma which was caused by a leak in this aneurysm. In

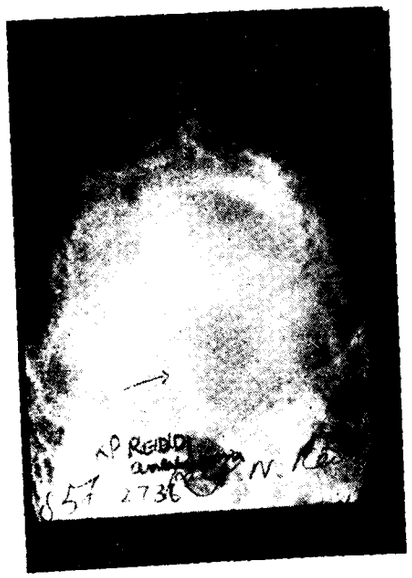

Fig. 16
K.P.R. Town's view of the skull. Arrow points to a circular calcified shadow in the wall of the aneurysm.

spite of repeated requests to get operated on, the patient refused. Three years later he was readmitted with symptoms of a typical frontal lobe lesion. Angiography was repeated and showed the aneurysm to be bigger in size. He again refused surgery. Two days later he expired.

This case shows how an aneurysm may cause an intracerebral haematoma. Rarely an aneurysm may be the cause of a subdural haematoma.

53 cases of intracranial aneurysms are analysed here.

Table 3

Age of onset of symptoms	Number of cases	Number of deaths	Mortality per-cent
10-20 ...	3	...	...
21-30 ...	14	10	71
31-40 ...	11	2	18
41-50 ...	11	4	36
51-60 ...	10	3	30
61-70 ...	4	1	25
Total ...	53	20	38

From the table it is obvious that the younger age group of 20-40 is more affected and the mortality is highest among the younger age group.

Table 4

Mortality

	Number of cases	Total deaths	Death within 48 hours	Death between 2-14 days
Cases with blood in C.S.F. ...	35	19	8	11
Cases without subarachnoid haemorrhage but with focal signs.	18	1 (2 years later)	...	...

It is also obvious that there is great risk of death in cases of aneurysms that manifest themselves with subarachnoid haemorrhage than with focal neurological signs alone.

The mortality table also shows the risk of recurrent haemorrhage within the first two weeks. This risk of recurrent bleed is an important factor to which special attention has to be drawn as this is the one factor that has stimulated advance of surgical treatment of intracranial aneurysms.

Symptomatology : When the aneurysm begins to bleed, symptoms are caused by the presence of blood in the subarachnoid space. This is chiefly manifested by the occurrence of very severe headache. Usually the onset of the headache is very sudden. The patient may lose consciousness for a few minutes or for a long time. Loss of consciousness depends upon the size of the bleed. 25 cases out of 35 showed at the onset, loss of consciousness after sudden headache. Headache persists and may spread from the temporal region or the frontal region to the back of the neck and also along the spine to the lumbar region. This headache and pain in the neck is due to the presence of blood irritating the meninges. The headache may persist for a few hours or more likely for a few days. In addition to the presence of pain and headache the patient may exhibit occasionally other neurological signs that may give a clue to the side of the subarachnoid haemorrhage. There may be third nerve paralysis or disturbance of vision on one side. There may also be presence of weakness of one limb or the other. In 16 cases out of the 35 of subarachnoid haemorrhage showed lateralising signs.

Occasionally an aneurysm may manifest itself solely by a focal neurological lesion when the leak is minimal. The risk of fatality is very little when the patient presents himself with focal neurological signs without subarachnoid haemorrhage. In such cases plain X-rays may show occasionally circular calcified areas suggesting an aneurysm. (fig. 17)

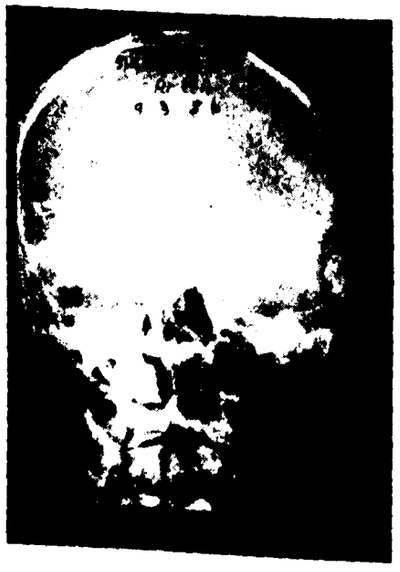

Fig. 17
Cross circulation test with compression of one carotid. *Note* the filling of the middle and the anterior cerebral arteries.

When any patient presents such focal neurological signs it is essential that the cerebral

vascular system is investigated for any aneurysm by cerebral angiography. In the 53 cases of aneurysm 18 cases presented primarily with focal neurological signs.

Table 5

Total number of cases with focal fits investigated ...	222
Number of cases of Intracranial aneurysm manifesting on focal fits	3—1.3%
Number of cases of Arterio-venous malformation manifesting focal fits ...	6—2.7%
Total number of cases of III Neuropalsy ...	54
Number of cases of intracranial aneurysm detected ...	10—18.5%

The following table shows the variety of neurological lesions met with.

Table 6

Symptoms of Intracranial Aneurysms

Serial Number	Symptoms at onset	Number of cases
1.	Headache	All the cases
2.	Loss of consciousness ...	25
3.	Hemiparaesis	16
4.	Focal fits	3
5.	Proptosis	2
6.	Ptosis	10
7.	Pulsating Exophthalmos ...	1
8.	Blindness of one eye with optic atropy	2

Diagnosis : Diagnosis of subarachnoid haemorrhage is chiefly made from the history of the case and is confirmed by lumbar puncture. In any case of sudden severe headache (occurring almost like a bolt from the blue) lumbar puncture is an essential procedure. Lumbar puncture reveals blood uniformly mixed with the cerebrospinal fluid. If the lumbar puncture is done later than 24 to 48 hours then actual blood may not be seen, in the cerebrospinal fluid but the fluid may be coloured yellow and may have a very high protein content. When the case is diagnosed as a subarachnoid haemorrhage further treatment has to be decided upon. If the patient is acutely ill, only conservative measures are adopted for the first 24 to 48 hours.

Cerebral angiography is absolutely essential in every case of subarachnoid haemorrhage. I prefer to wait at least two or three days after the onset of haemorrhage before advising a cerebral angiogram for the reasons mentioned below. When an aneurysm bursts there is intense vasospasm of all the vessels of the brain, tending to minimise the bleeding and to favour clotting. This vasospasm may be very intense and is a factor in causing unconsciousness which is also a blessing for the patient who has otherwise a terrible headache. If the vasospasm involves the brain stem vessels, death may occur. This vasospasm perists for 48-72 hours. This has to be borne in mind because a cerebral angiogram done within the first 72 hours may not fill all the cerebral vessels and the aneurysm may be missed. The vasospasm also prevents proper surgery when attempted within 48 hours.

Cerebral angiography is necessary to diagnose the site of the haemorrhage so that further definitive surgical treatment may be decided upon. Carotid angiography is done on both sides. If there are any focal neurological signs the angiogram is done on the corresponding side. If on finishing the angiogram on one side no aneurysm is visualised then the other carotid angiogram is done. If bilateral carotid angiography does not show any aneurysm, it may be necessary to do a vertebral angiogram. Injection of one vertebral is sufficient to show both sides. During angiography a cross circulation test is done to check the efficacy of collateral circulation from the opposite side. This is done by compressing one common carotid artery while the dye is being injected into the other and finding out whether the opposite anterior cerebral or the middle cerebrals artery fills. (Figure 18). After this investigation is completed treatment is decided upon depending upon the site of the aneurysm. Without going into elaborate details of treatment it may be pointed out here that in any aneurysm of the internal carotid artery upto its bifurcation, carotid ligation in the neck may suffice to give a good chance for the aneurysm to heal. For treatment of aneurysm of the circle of Willis

it is generally agreed that a direct attack on the aneurysm is a desirable procedure though some centres still practise carotid ligation. Carotid ligation for intracranial aneurysm is

Fig. 18

G.T.H. Angiogram arterial phase. *Note* the tumour in the occipital region. *Note* also the anastomosis between branches of the posterior cerebral artery and the occipital artery.

an accepted and useful procedure. It diminishes the blood flow through the aneurysm thus diminishing the risk of rupture and creating favourable conditions for clotting of the aneurysm. Many centres still practise this procedure. There are many pitfalls in carotid ligation. It is very essential to make sure in every case, where ligature of the carotid artery is decided upon that there is sufficient collateral circulation from the opposite side. This can be done by compressing the opposite carotid artery when doing a cerebral angiogram. In addition Matas test of compressing the artery for about 20 minutes, watching for development of abnormal neurological signs on the opposite limbs is also a useful procedure.

Sometimes in spite of all these tests, hemiparesis may develop after carotid ligation. In one case (M.R.) it was necessary to release the carotid ligature half an hour after application as the patient developed hemiparesis. In another case the patient developed slight aphasia 48 hours after carotid ligature in spite of a good collateral circulation demonstrated by angiography. These are due to anomalies in the circle of Willis which are commonly associated with the aneurysm. But obviously carotid ligation does not deal with the problem completely. The ideal treatment would be obliteration of the aneurysm by a direct approach. A suitable osteoplastic flap is turned and the aneurysm exposed. The neck and the sac are defined and the aneurysm is clipped. If clipping is not possible, without endangering the main arterial supply to the distal portions, muscle may be wrapped round the aneurysm. Attempts are also being made to form a rigid coat around the vessel with the aneurysm, by pouring around them a polymetised solution which sets and becomes hard. In certain situations like the distal portion of the anterior cerebral, it may be possible to clip the main vessel itself without endangering brain function if the circulation from the opposite side is good.

Table 7

Sites of aneurysms

Serial Number	Sites of aneurysms	Number of cases
1.	Internal carotid (infraclinoid)	7
2.	Internal carotid (supraclinoid)	3
3.	Anterior cerebral ...	1
4.	Middle cerebral	2
5.	Posterior cerebral	1
6.	Basilar	1

The results of surgical treatment of aneurysms has been followed up very carefully by many centres and it has been found that surgical treatment definitely offers a better chance of a complete cure than conservative treatment. To summarise the experience of many neurosurgical centres regarding prognosis it may be said—

(1) When complete cerebral angiography does not reveal the presence of any aneurysm it is found that the prognosis is much better with conservative therapy than when conservative therapy is adopted in cases in which a definite aneurysm is demonstrated.

(2) That there is a great risk of recurrent haemorrhage developing within the first eight weeks (See Table 4).

(3) The results of conservative treatment have shown that if a patient escapes the recurrent bleed within the first eight weeks, the chance of further bleed is slightly less. But within the first eight weeks the chance of a recurrent haemorrhage is indeed great. About one-fourth of the cases of subarachnoid haemorrhage go through life without much further trouble. The remaining 5 per cent of these cases run a risk of sudden fatal haemorrhage. In such circumstances surgery has come as a definite life saving measure.

The present trend in the treatment of ruptured intracranial aneurysms is turning in favour of surgery. It has been proved that surgery done after three days of the bleed prevents quite a large number of fatalities from recurrent bleeding that occurred within the first two weeks. Surgery is now safer with hypothermia. Conservative treatment has nothing to offer in this period when fear of recurrent haemorrhage exists.

What of the future ? Is it possible to prevent deaths that occur within the first three days of haemorrhage. As pointed out earlier, diagnosis and localisation of the aneurysm is rendered difficult during the first 48 hours because of the intense protective vasospasm of all the cerebral vessels. This vasospasm makes early surgery also difficult because of cerebral anoxia. In addition, there is an associated cerebral oedema soon after the subarachnoid haemorrhage. It is also possible that the associated intracerebral haematoma adds to the complications. Till now surgery within first 48-72 hours has not appreciably diminished the mortality in this period, except for the fact that in the surgically treated series the aneurysm has been definitely cured whereas in the conservative group the aneurysm is there, still waiting to bleed. Papaverine administration has helped to overcome the vasospasm. With advances in hypothermia, it may be possible in future for emergency surgery to be done in early bleeding aneurysms with more successful results than now.

The Neurosurgeon has got much to be thankful for to his colleagues, the radiologist for accurate diagnosis and the anaesthetist for facilitating accurate surgery. Since the advance of hypothermic anaesthesia, more direct attack on the aneurysms is possible. Hypothermia allows cutting off of brain circulation for some minutes if necessary. During the dissection of the aneurysm, the aneurysm may rupture. It may be necessary at this stage to temporarily occlude the parent vessel. In such circumstances the risk of lack of blood supply to the brain occurs and a permanent neurological deficit may be precipitated. But this has been avoided by operating under hypothermia. The patient's temperature is brought down from the normal of about 36 to 27 C. At this temperature the brain is able to withstand anoxia for a much longer time and this facilitates the surgeon to isolate the vessels and the aneurysm calmly and without any hurry and then clip its neck. It is good practice to expose both carotid arteries in the neck as a first step in the operation. A clamp is loosely applied round the artery on the affected side and then tightened when necessary. This is a more reliable method than digital compression of the artery. In our experience hypothermia has been a boon not only in the treatment of aneurysms but also in the treatment of advanced intracranial tumours.

(3) *Vascular tumours* : In the discussion on intracranial vascular lesions, I have included also vascular tumours that occur inside the cranium. The important ones are the vascular meningiomas and haemangiomas occurring in the brain. In a series of 45 meningiomas treated in the General Hospital, Madras, 8 cases were very highly vascular tumours. The problems connected with the treatment of such lesions are almost identical with the problems connected with the arteriovenous malformations with the addition that these meningiomas also present the added difficulty of increased intracranial tension. Unfortunately these cases often come to the Neurosurgeon at a very late stage in our country. Hence these tumours acquire extra blood supply not only from the internal carotid system but also from the external carotids. They also cause severe pressure effects on the skull and the brain. In the days when there were no additional aids like hypotension or hypothermia these enormous meningiomas could not be removed successfully. In spite of replacement of the blood and all attention being paid to the neurosurgical haemostatic technique it was not possible to remove these enormous tumours successfully. But with hypotension and hypothermia 9 cases of huge vascular meningiomas have been removed. One important lesson that has been learnt in the operation for the removal of such huge meningiomas is

that in spite of hypotension it is necessary to start replacing early in the operation the blood that is being lost by the patient. With hypothermia the problems are easier as hypothermia by itself reduces the bleeding and also considerably reduces the intracranial tension (Rajagopal) 7. It also helps by enabling the surgeon to expose the carotid or the vertebral arteries and to occlude them if necessary for as long a period as ten to fourteen minutes when dealing with an enormous tumour.

Mr. G. T. H. aged 30 years was admitted with a history of headache, vomiting and blindness. A diagnosis of an occipital tumour was made and a cerebral angiogram done (fig. 19)

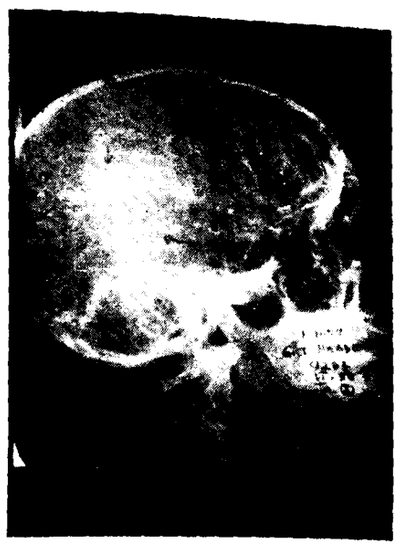

Fig. 19
G.T.H. Capillary phase showing the tumour.

The pictures show the enormous vascularity of the tumour. They also show the communication between the internal and the external carotid systems namely the posterior cerebral and the occipital arteries. The patient was operated upon under hypothermia in two stages. During the operation one carotid artery was occluded for over 90 minutes and the opposite one occluded simultaneously for ten minutes. A huge meningioma attached to the falx cerebri, the tentorium the straight sinus and the transverse sinus was removed. But for hypothermia it would not have been possible to remove this tumour.

Haemangiomas

Two cases of haemangiomas of the skull are also included in this series. These are rare tumours and only about 50 cases have been reported till now in world literature. These tumours arise in the skull bone and slowly begin to grow inwards as well as outwards. The patient may present either with symptoms of focal irritation of the cerebral cortex or with a tumour in the scalp or both.

On examination the tumour is found to be vascular, warm and pulsating. Due to bone reaction to this tumour new bone is formed around the periphery and this may be felt as a saucer shaped elevation all round at the base of the tumour. The diagnosis is made by the X-ray appearance. Radiating spicules of bone, with a typical sun-ray appearance are characteristic of haemangiomas of the skull (Figure 20).

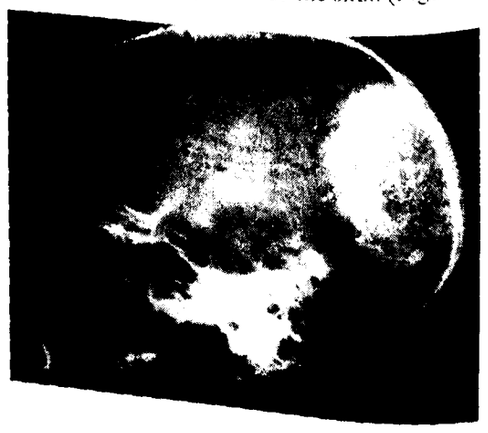

Fig. 20
N.S. 9051 Hæmangioma of the skull lateral view showing the typical sunray appearance.

In the interspace between the radiating spicules of bone, the haemangioma communicated with the intracranial space. Complete removal of the tumour is possible by excising the entire affected bone en masse. The defect is grafted by polythene or acrylic resin. In huge tumours hypotension is helpful for surgery, as the tumours acquire plenty of blood supply from the meninges and the scalp.

Haemangiomas of the brain occur commonly in the cerebellar region. They may also occur in other portions of the brain and are usually symptomless. Cerebellar haemangiomas form a definite entity and resemble cerebellar

tumours in their symptomatology. They usually occur in the cerebellar lobe but may also occur in the vermis. No special problems are associated with these vascular tumours except the following—

(1) Cerebellar haemangiomas may be associated with cysts in the kidney and the pancreas and then is known as Lindau's disease.

(2) Polycythemia has been reported in association with cerebellar haemangiomas.

Sturge Weber Syndrome : Cases of Sturge Weber Syndrome have been included in this study. All these cases have been completely investigated by pneumoencephalogram and carotid angiogram (Ramamurthi B and Mani K. S.)[8]. Sturge Weber Syndrome is an association of haemangiomas of the face with focal neurological deficit and an associated calcification of the cerebrum chiefly in the occipital lobe. The wavy parellel calcification seen in the cortex is not due to calcification in the blood vessels, but due to the deposit of calcium in the cortical layers.

The problem of internal carotid thrombosis is dealt with in a separate paper.

Conclusion : The Neurosurgeon has much to be thankful for to the Radiologist for better diagnosis and to the anaesthetist for better treatment. Without the willing and devoted services of a good anaesthetist such surgery of intracranial vascular lesions is impossible. When such aids are available, the Neurosurgeon can look forward confidently into the future for better and earlier amelioration of the afflictions of one of the most intricate systems of the human body.

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AORTOGRAPHY

BY

R. NIGAM, A. M. GHOOI AND S. V. TUNGAR, NAGPUR

This paper reviews our observations on Aortography carried out by the translumbar and retrograde catheterisation techniques for the diagnosis of lesions of the Aorta and its larger branches, lesions of the kidneys, and obliterative vascular disorders of the lower limbs. Forty cases have been studied so far.

The pioneer of lumbar aortography was Reynaldo dos Santos, who reported a series of 300 cases in 1929 (dos Santos, Lorner and Caldos. 2). There was an element of danger in his procedure because the contrast medium was 80% sodium iodide. If this went into the superior mesenteric artery in any quantity, it caused thrombosis of the vessel and gangrene of the bowel. The investigation was repeated on dogs by Henline and Moore in 1936 with the same contrast medium with more disastrous results. It was with this background of danger that Nelson(4) and later Doss(1) (Doss, Thomas and Bond) and Wagner (7) had to contend and it showed considerable courage that they persevered in their efforts. The use of organic iodine preparations instead of sodium iodide has abolished nearly all the dangers. Griffiths (3) reported the first series of cases in England using diodone 70%. Riches (5) reported 200 cases of aortography by the translumbar route, especially for lesions of the kidney. There was no mortality in his series and only a few minor complications were observed. Smith, Rush and Evans (6) reported a series of 1,000 aortic punctures for arteriography without fatality and without significant morbidity.

Diagnostic Value

The procedure, correctly speaking, is an arteriography performed by the translumbar or the retrograde routes for visualising the aorta and its larger branches. The pattern of renal blood vessels can be demonstrated with significant alterations in their pattern in benign or malignant lesions of the kidney. It is not always possible to differentiate these lesions accurately by retrograde or intravenous pyelography. Thrombotic lesions and aneurysms of the aorta and its larger branches can be well demonstrated. It is of great help in diagnosing retroperitoneal tumours, particularly in distinguishing renal swellings from other tumours.

Routes

There are two routes of approach to the abdominal aorta, the lumbar and the femoral. In the lumbar approach the needle is entered below the 12th rib about six inches from the midline. It is directed into the aorta by the side of the first lumbar vertebra.

In the femoral approach, as described by Pierce, a polythene catheter is passed via a special canula into the femoral artery and is guided up the artery to a various extent in the abdominal aorta. We have preferred the translumbar approach as it is easier, quicker and less prone to failure or to complications, than the femoral route.

For visualisation of the thoracic aorta we have adopted either the transcarotid or the transbrachial routes.

Dangers

Translumbar aortography as described by dos Santos was associated with a serious risk of thrombosis of the superior mesenteric or renal arteries, and with associated complications of mesenteric infarction or anuria. However with the use of organic iodine compounds such complications are rare. Theoretically it is possible to cause an extravasation of blood at the site of puncture causing postoperative discomfort. We have observed this occasionally in a difficult case but the symptoms disappeared within a few days. Most of our cases were symptom free within 24 hours with only slight tenderness as a result of the needle puncture. We have had no case of anuria as reported by Riches. The sensitivity to iodine was determined in every case, the investigation being avoided in patients sensitive to iodine.

Causes of failure

The main cause of failure is a faulty position of the needle tip. After practice on the cadavre and with some clinical experience, the abdominal aorta is tapped with the greatest of ease. One point of detail as observed by one of us (R.N.) was the value of passing the needle with a stilette in position at the first instance. After the appropriate position on the body of the first lumbar vetebra has been reached, the stilette is withdrawn and the needle passes easily into the aorta. The correct position of the needle tip is indicated by the free flow of blood.

Equipment

This essentially consists of a 30 c.c. syringe with luer lock attachment, a polythene tube of 2mm. bore attached firmly to a three way stopcock. A six inch 18 gauge needle with a keen edge and with a stilette. The iodine compound used by us was pyelocil 50% and 75% strength. Approximately 30 cc. was used in a single case. (Fig. No. 1).

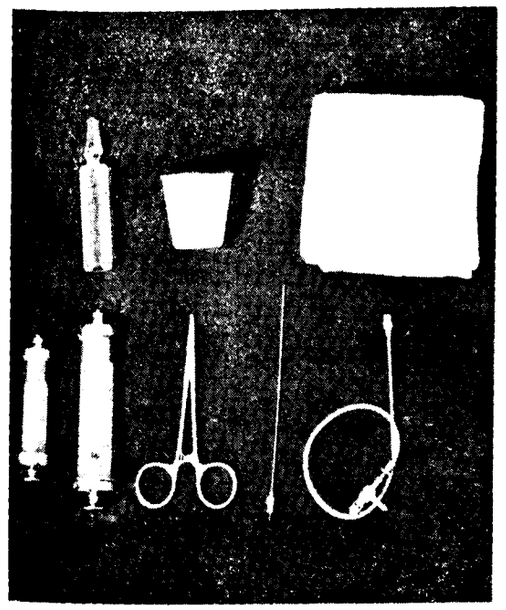

Fig. 1
Equipment used.

Technique

The patient is prepared by an enema in the morning and nothing is taken orally for about eight hours prior to the test. An injection of 100 mgms. pethidine with atropine is given an hour prior to the test. The patient is placed prone on the X-ray table. In the beginning a scout X-ray film was taken to check the position of the patient. This precaution was found unnecessary later on. A point four fingers breadth lateral to the midline is taken on the left side of the spine of the first lumbar vertebra and is prominently marked (Fig. No. 2). The needle is passed in an upward and inward direction at an angle of 45 degrees to the surface till the point of the needle is in contact with the body of the first lumbar vertebra. The stilette is withdrawn and the needle advanced till it just passes the side of the body of the vertebra. The needle as it enters the aorta, will suddenly be released from resistance it has encountered from the aortic wall, much as a needle entering the subarachnoid space. The proper position of the needle is revealed by a free flow and frequently a spurt of blood. Immediately after the needle enters the aorta, it is attached to the polythene tubing by luer lock attachment and a solution of citrated normal saline is slowly injected till the radiologist is ready to take a skiagram. The syringe full of pyelocil is now attached to the polythene tubing and the drug rapidly injected. Polythene tubing with a transparent wall is valuable. It reveals a regular pulsatile column of blood indicating the correct position of the needle tip. An X-ray plate placed under the patient is exposed when 15 cc. of the drug has been injected and the remainder is in the process of injection. The usual timing of injection was three to four seconds. In patients where the abdominal aorta and the femoro-popliteal vessels had to be visualised, a second X-ray plate, placed lower down, from the thigh to

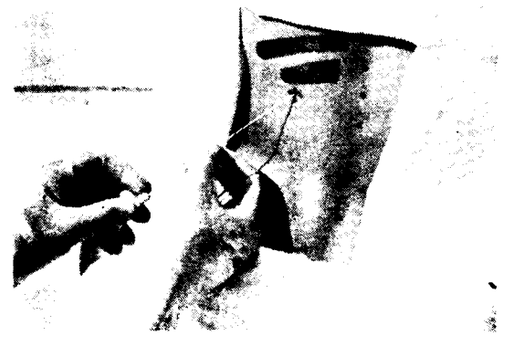

Fig. 2
Procedure of injection.

the middle of the calf, was exposed a few seconds after the first plate. An X-ray machine with two tubes is employed for this work. By proper team work with two radiographers this can be easily accomplished. We were not able to demonstrate the venous filling phase, owing to the non-availability of a rapid film changer. The needle, after the X-ray exposures, is rapidly removed, and the patient turned on his back. The whole procedure taking approximately 3 to 5 minutes and requires 0.25 gm. of pentothal anaesthesia.

For retrograde aortography of the thoracic aorta, a polythene catheter 1mm. bore was passed down along the brachial or the common carotid artery through a small nick in the arterial wall. The position of the catheter was determined by the length to which it had been pushed in. Pyelocil was rapidly injected and films were exposed. This observation was carried out under I. V. Pentothal anaesthesia. The artery was later repaired with a few sutures of 5/0 arterial silk. No untoward complications were observed. In the brachial route, the radial pulse, on the operated side, was less prominently felt. In one case a brachial plexus block and local procaine infiltration was employed. The radial pulse was normal on the following day.

Observations

The majority of our observations have been those of translumbar aortography for obliterative vascular disease, or lesions of the kidney. Only four cases of retrograde aortic catheterisation were observed for lesions of the thoracic aorta.

(*a*) *Normal Aortogram :* Three cases are illustrated here. Fig. No. 3 shows normal visualisation of the kidneys along with the abdominal aorta. In fig. No. 4 one kidney has been visualised along with the aorta. In this case the needle tip was at the level of the right renal artery. Fig. No. 5 shows visualisation of the aortic bifurcation.

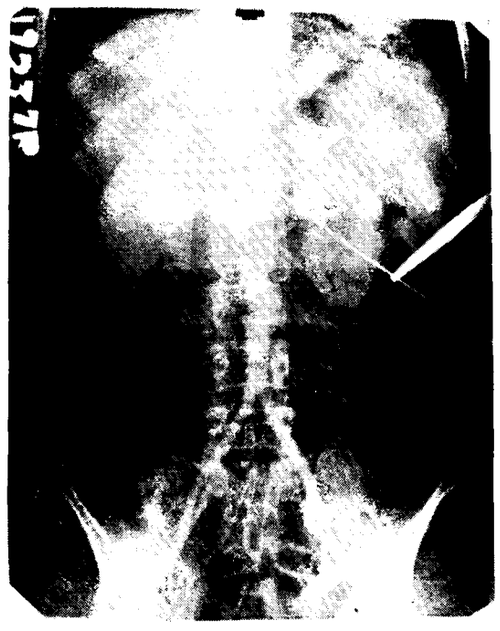

Fig. 3
Normal Aortogram
Both kidneys visualised.

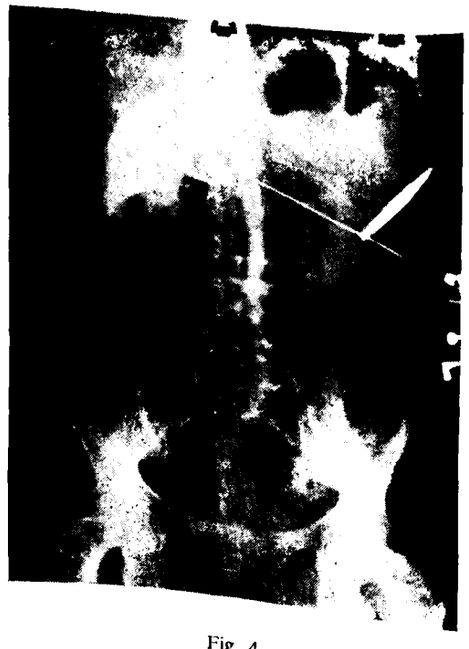

Fig. 4
Normal Aortogram
Right kidney visualised.

Fig. 5
Normal Aortogram Aortic bifurcation.

(*b*) *Obliterative vascular lesions of the lower limbs :* It has been our observation that in patients with obliterative vascular lesions of the lower limbs, a thrombotic lesion in the common or the external iliac arteries was present in association with a similar lesion in the popliteal arteries. This is illustrated in fig. No. 6 a case of gangrene in the left leg as a result of a block in the left popliteal artery. His aortogram showed, in addition, a block in the right common iliac artery. Fig. No. 7 illustrates a segmental block of the right common iliac artery. This patient was subsequently operated upon and a by-pass of the obliterated segment was achieved by a dacron arterial graft.

(*c*) *Kidney :* (1) *Ectopic kidney.* This is well illustrated in figs. Nos. 8 and 9. The abnormal origin of the renal vessel is clearly visualised.

(2) *Aberrant renal artery.* Fig. No. 10 shows an aberrant vessel supplying the lower pole of the left kidney. This was an accidental observation in a case of obliterative disease of the iliac artery.

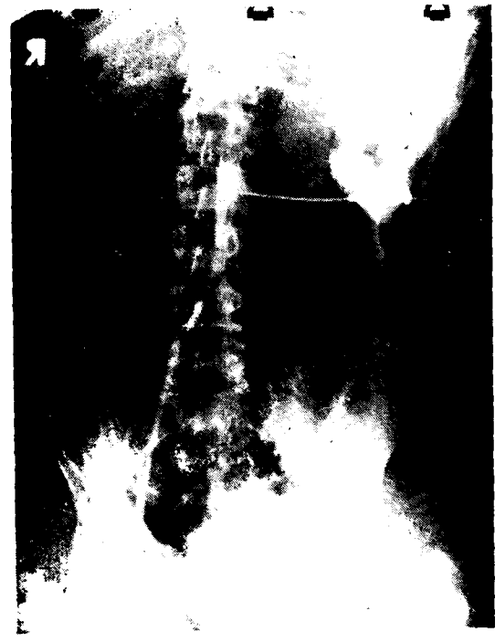

Fig. 6
Obliteration of left common iliac artery.

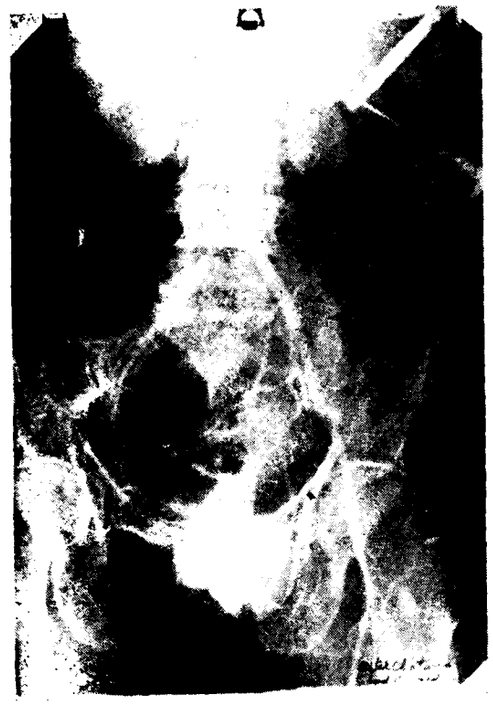

Fig. 7
Segmental block of right common iliac artery

Fig. 8
Pelvic kidney Lt. (Pyelogram)

Fig. 10
Aberrant renal artery Lt.

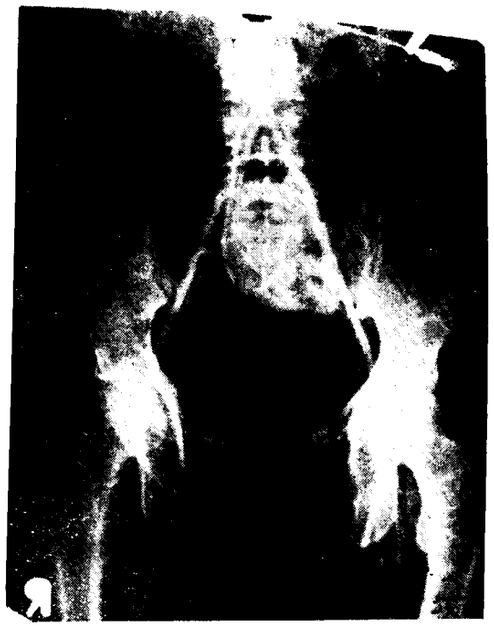

Fig. 9
Pelvic kidney Lt. (aortogram) (same case as No. 8)

Fig. 11
Bilateral Hydronephrosis

(3) *Hydronephrosis.* Fig. Nos. 11 and 12 show the aortograms of two cases of hydronephrosis due to calculi. The comparative avascularity in the kidney region with displacement of the aorta by the hydronephrotic kidney is demonstrated.

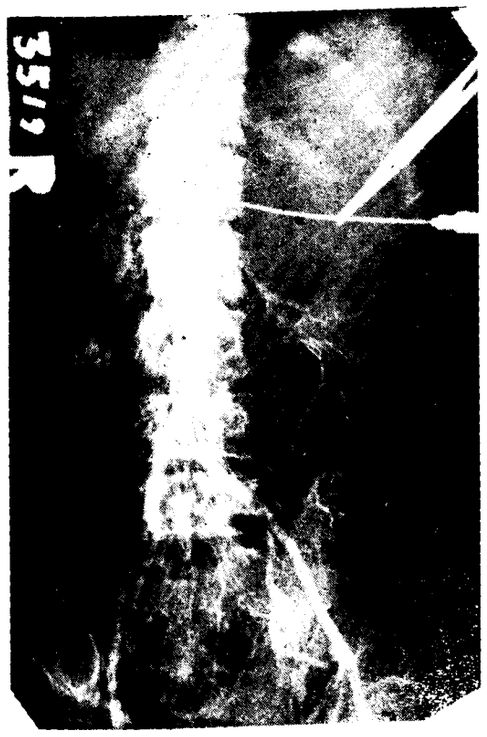

Fig. 12
Bilateral Hydronephrosis

(4) *Polycystic disease.* A case of bilateral disease showing an advanced lesion on the left side, and with early cystic changes on the right side is shown in fig. No. 13.

(5) *Kidney tumours.* A case of Grawitz tumour of the left kidney Fig. No. 14 very well illustrates the distortion of the pattern of the renal vessels with pooling of the dye in the kidney substance.

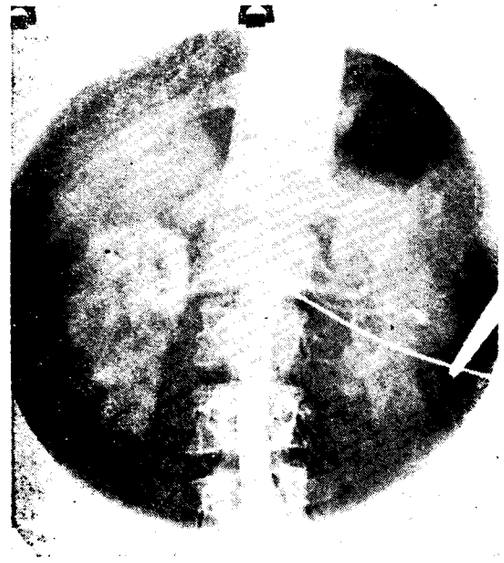

Fig. 13
Polycystic disease kidneys.

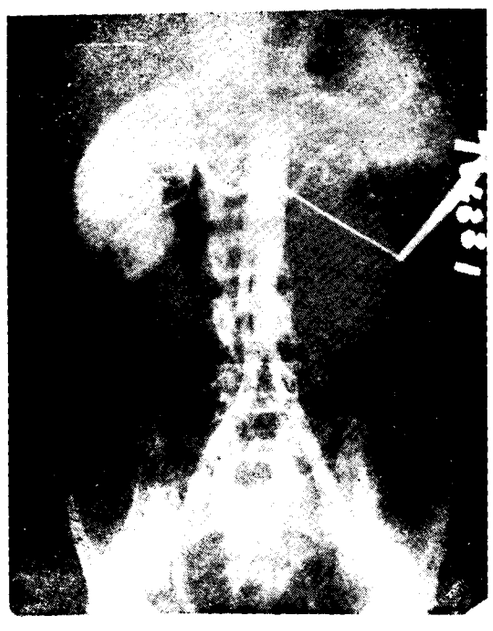

Fig. 14
Von Grawitz Tumour Lt. side.

(*d*) *Vascular tumours in the proximal part of the femur*. Aortogram in a case of osteosarcoma of the upper 1/3rd of the left femur (fig. No. 15) shows increased vascularity and displacement of the femoral artery.

Fig. 15
Osteosarcoma left femur.

Fig. 16
Aneurysm ascending aorta.

(*e*) *Retrograde aortogram*. These were performed in two cases of aortic aneurysms and in two cases of innominate aneurysms. Fig. No. 16 shows the aortographic findings in a case which presented a pulsatile tumour low down in the chest on the right side, consistent with an aneurysm. It was difficult to find out, by routine radiography, as to whether it was arising from the ascending or the descending aorta. Retrograde aortography, through the right common carotid artery, revealed it to be a case of an aneurysm of the ascending aorta.

Conclusion

Translumbar aortography is a relative simple and harmless procedure. It provides valuable information about obliterative vascular lesions in the aorta and its larger branches. It also helps to demonstrate lesions of the kidney and in differentiating benign from malignant conditions. It is also valuable in demonstrating the position of a kidney and its blood supply. No untoward complications were observed in over forty aortographic procedures undertaken by us, by the translumbar route.

Retrograde aortography was employed for the thoracic aorta in four cases. There were again no untoward symptoms in these patients.

Acknowledgment

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SIMPLE TUMOURS OF SMALL INTESTINE AND CAECUM CAUSING INTUSSUSCEPTION

(A REPORT OF SIX CASES)

BY

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H. S. BAWA, AMRITSAR

Simple tumours of the small intestine are uncommon lesions which are rarely diagnosed clinically, as no single surgeon is likely to come across more than a few cases over a number of years. Strohl and Diffenbaugh (1957) in a five year review of admissions to St. Luke's Hospital, Chicago, found 12 instances of tumours of small intestine, of which 4 were benign. Most of the tumours remain small and asymptomatic during the lifetime of the patient and are encountered as an incidental finding at laparotomy or at autopsy. According to River et al (1956) benign neoplasms of small gut are found 15 times as often, among 1,000 autopsies, as among a like number of surgical specimens. However some of them produce pain, obstruction, haemorrhage or other symptoms. Intussusception is the most common type of obstruction produced by simple tumours of small intestine and is often of the recurrent type. It is of interest, therefore, to report 6 cases of simple tumours of small gut and caecum, all of which produced intussusception and were successfully treated by resection. Three of these tumours were haemangiomas while the other three were fibromas.

River et al (1956) in their excellent article reviewed 1399 benign neoplasms of small intestine reported in literature upto 1954, including 20 of their own, but excluding those from the Mayo Clinic. Out of these 127 were angiomas and haemangiomas and 163 fibromas. The commonest simple tumours were adenomas. the next common being lipomas. Olson et al (1951) in a review of 77 benign neoplasms of small intestine collected from the specimens and records of the Mayo Clinic from 1911 to 1942 found that eleven of these were fibromas while only 4 were haemangiomas.

Gentry et al (1949) classified benign vascular lesions of the small gut as follows :

A. Telangiectasis (Hereditary or non-hereditary),

B. Haemangiomas.
   (*a*) Capillary Haemangioma (Simplex, mostly single).
   (*b*) Mixed capillary and Cavernous haemangioma.
   (*c*) Cavernous haemangioma :
      (1) Multiple phlebectasia (Small cavernous).
      (2) Single polypoid (Simple cavernous).
      (3) Diffuse expansive (Single contiguous).
      (4) Diffuse expansion (Multiple non-contiguous).

They reviewed 36 cases of single polypoid cavernous haemangioma from literature and reported 14 of their own. Capillary haemangioma was the next common vascular lesion in these series, reviewing 12 cases from literature and adding 6 new ones. Hensen (1948) collected 11 cases of polypoid cavernous haemangioma and 8 cases of capillary haemangioma in literature and reported one new case. Capillary haemangioma of caecum is still rarer. Gentry et al (1951) recorded 2 cases described in literature and one of their own which was found as an incidental finding at autopsy.

Two of the six benign tumours reported here were haemangiomas of small intestine, while the third haemangioma was in the caecum. According to the above classification, one was a single polypoid cavernous haemangioma (Case V) and others were capillary haemangiomas of small gut (Case I) and of caecum (Case II). The remaining three tumours were fibromas of small gut. One of them had undergone a myxomatous degeneration (Case III) and another showed a hyaline change (Case IV).

Simple tumours may be intraluminal, when they may become pedunculated, or extraluminal which may grow to a large size and contact adhesions. They may undergo cystic

S. No. and Name	Age	Sex	Admitted with history of acute Obstruction: Pain	Vomiting	Constipation	Distension	History of sub-acute Obstruction	Past Illness: Pain	Constipation	Ball of wind	Bleeding per-rectum	Physical Finding: Palpable lump	Visible peristalsis	X-Ray Findings: Barium meal	Barium enema	Operative Findings	Operative procedure	Histopathology	Result
1. P.K....	26	F	+	+	+	+	—	—	—	—	—	—	+	...	...	...	Ileo-ileal intussusception.	Reduction. Resection and end to end anastomosis.	Capillary Haemangioma.	Cured.
2. J.S. ...	30	M	+	—	—	+	+	+	—	—	—	—	+	...	...	Obstruction at hepatic flexure.	Do.	Do.	Fibroma	Do.
3. F.S. ...	25	M	+	—	+	+	+	+	—	—	—	—	+	...	...	Filling defect ascending colon.	Do.	Do.	Fibroma undergoing myxomatous degeneration.	Do.
4. B.D....	27	M	+	+	+	+	—	—	+	—	—	—	+	Distended coils of small gut.	...	...	Ileo-colic Intussusception.	Do.	Fibroma with a hyalane change.	Do.
5. S.K. ...	28	F	+	+	+	+	+	—	—	—	—	—	+	...	Distended coils of small gut.	...	Ileo-ilial Intussusception.	Do.	Cavernous haemangioma.	Do.
6. A.D....	29	F	+	—	+	+	—	—	—	—	—	—	+	Distended coils of small gut.	...	Filling defect in ascending colon.	Ileo-colic Intussusception.	Right Hemicolectomy.	Capillary Haemangioma.	Do.

Note—F Female. M — Male.

calcarions, hyaline or myxomatous degeneration. Five of the six tumours reported in the present series were intraluminal.

Many of the benign neoplasms of small intestine do *not* produce symptoms. The severity of symptoms depends upon the site, size and relationship of the tumour to the bowel wall, the intraluminal type producing the symptoms earlier. Pain, constipation sometimes alternating with diarrhoea, bleeding (small and intermittent or copious), palpable mass mostly due to intussusception or symptoms of sub-acute or acute intestinal obstruction may be present. Even when patients present with acute obstruction, a history of previous mild pain, constipation, anorexia or a ball of wind is usually forthcoming.

The commonest complication of simple tumours of small intestine is intussusception. It was present in 627 of the 1399 cases collected by River et al (1956). Various reports on intussusception in adults show that simple tumours of small intestine were the commonest cause of this form of obstruction (Bosworth and Stein, 1947 : Roper, 1956). It is important therefore to keep the possibility of finding such a cause in mind, because after reduction of intussusception the tumour may be overlooked necessitating a second or even multiple operations. The intussusception may reduce spontaneously and recur at intervals, producing symptoms which may vary from the classical picture of intussusception in infants so much that a definite diagnosis is rarely made. Haemangiomas of small intestine are said to produce intussusception less often than other neoplasms (Rankin & Newell, 1937 ; Butler et al, 1951). Other complications of benign neoplasm of intestine are intestinal bleeding, peritonitis, necrosis, degeneration and malignant change in polyposis with melanin deposit.

X-ray examination even with barium meal, usually fails to visualize the growth, though some successes have been reported in the literature. Straight roentgenogram of abdomen in patients with intussusception reveal distended coils of small gut and in the ileocolic type may show an absence of gas in the right iliac fossa. In this type a barium enema will confirm the diagnosis by showing the pinzer or coiled-spring appearance. The tumour causing the intussusception may or may not be visualized.

Three of our cases (Chart I) were admitted to our surgical unit with a history of sub-acute obstruction going on to acute obstruction. The other three were admitted with signs and symptoms of acute intestinal obstruction of a short duration. Three cases, all of fibromas of small intestine, gave a previous history of pain or constipation of 2 months to 6 years duration. In one of these cases pigmentation of skin and subcutaneous neurofibromata were present. Intussusception was found on exploration in all the 6 cases. It was of ileo-ileal type in 4 cases. In the other 2 it was of the ileo-colic variety. X-ray investigations with barium meal or barium enema were carried out in 4 of our cases. In one case the investigation showed dilated coils of small gut. The others either showed a filling defect or complete obstruction to the flow of barium. In 2 cases only was there a suggestion of intussusception.

Case No. 1

P.K., 26 years, housewife, admitted on 16th December 1952.

History : About 2 days before admission the patient felt acute pain in the abdomen. The pain became more severe after a few hours and she started vomiting profusely. At first the vomits consisted of gastric contents but later she started vomiting black, bad smelling fluid. She did not pass faeces or flatus for 2 days. Then gradually, she developed some distension in the central portion of the abdomen. No history of diarrhoea, dysentery or bleeding.

General examination : Moderately nourished, anxious facies, eyes sunken, not anaemic, tongue dry and coated, pulse 90 min., temp. 98.2 F., B.P. 130/80.

Local examination : The abdomen was distended centrally with visible peristalsis. On palpation the abdomen was tender all over. No mass could be palpated. Rectal examination revealed a mass of distended coils of gut which could be felt anteriorly. There was no blood or mucous on the examining fingers.

Laboratory investigations : Hb. 12.47 grams.%, Total Leucocyte count 14,000 c.mm., Neutrophils 79%, Lymphocytes 21%. Bleeding time 2 minutes, coagulation time 3.5 minutes.

Operation : 16th December 1952. On laparotomy an ileo-ileal intussusception caused by a tumour was detected. The apex was about 30 cms. from the ileocaecal junction and the neck was located the same distance farther away. The intussusception was easily reduced and on reduction four patches of sloughs were found near the apex and the neck. Resection of the

part with end to end anastomosis performed and the wound closed in layers.

Pathological report : The specimen consists of 9" of opened up ileum with a spherical tumour-like mass, dark grey in colour, size of diameter 3 cm., circumference 8 cm., protruding into the lumen of the ileum and arising from the mucosal surface of the antimesenteric side of the gut wall (Fig. 1). Firm in consistency. Section of the tumour showed the cut surface to be solid and dark grey in colour.

Fig. 1

Case I. Excised portion of the ileum with an intraluminal pedunculated haemangioma in its distal part.

Histology : Section shows histology of capillary haemangioma (Fig. 2).

Post-operative period : Developed retention of urine and had to be catheterized repeatedly. Discharged on 5th January 1953 as cured.

7th September 1953 : Reported for follow-up nearly 8 months after the operation, had no bowel complaint.

Case No. 2

J.S., 30 years male, was transferred to East Surgical Ward of V.J., Hospital, Amritsar on 14th January 1955 from medical section where he had been admitted earlier

Fig. 2

Same case as in Fig. 1. Microphotograph, low power showing structure of a capillary haemangioma.

for investigation of sub-acute intestinal obstruction. The complaints dated back to 6 years before admission. He used to get colicky attacks of generalised pain in abdomen, coming on after 5 or 6 days and lasting for an hour or so. Since 1¼ months before admission he started getting pain every day and there was increasing distension of abdomen. There was no vomiting or rise of temperature. At no time did he get absolute constipation.

General examination revealed mild degree of anaemia in an emaciated patient. There were multiple patches of pigmentation on the back with a few neuro-fibromas. Abdomen was greatly distended in the central area. Visible pesistalsis could be seen. P.R.—No abnormality detected.

Haemoglobin was 9.4 Gm.%. Total Leucocyte count 8,600/c.mm., Differential Leucocyte count—73/26/1/0. Urine examination showed no abnormality. B.S.R.—4 mm. first hour Westergren. Radiogram after Barium enema revealed obstruction to the flow of barium at Hepatic flexure. Dilated coils of small gut seen in the same X-Ray (Fig. 3).

Laparotomy was undertaken on 25th January 1955, through a right paramedian sub-umbilical incision. On opening the peritoneum huge dilated coils of small gut were seen. At numerous places the serous covering over the distended coils had given way, exposing the muscle layers covered by filmy exudate. These dilated loops were traced down to an ileoileal intussusception about 2 feet from the ileocaecal valve. There were no vascular changes and the intussusception was reduced easily, when at its apex a firm growth projecting into the lumen was palpable. About four feet of the gut, including the growth was resected and end to end anastomosis done in 2 layers. The laparotomy wound was closed in layers without drainage.

Fig. 3

Case II. Roentgenogram after barium enema showing obstruction to flow of the opaque medium at hepatic flexure.

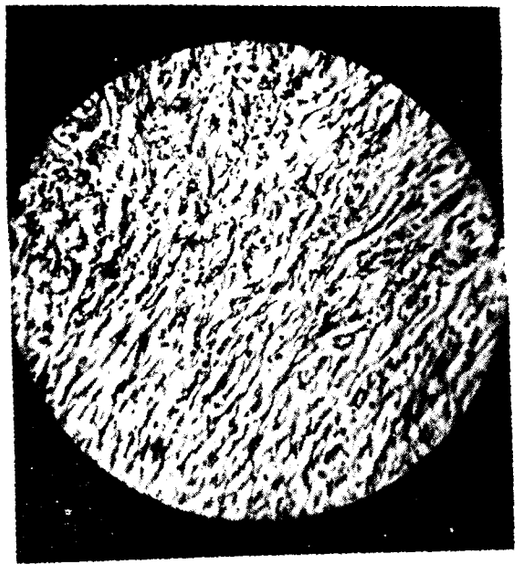

Fig. 4

Same case as in Fig. 3. Microphotograph, high power showing structure of a fibroma.

Pathological report : A 10 c.m. segment of markedly dilated small intestine bearing a dark friable growth with pedicle. Dimension of the growth 6.5 3.5 1.5 c.ms. Cut section shows haemorrhagic and pale areas.

Histology : Fibroma. (Fig. 4).

The patient made an uneventful recovery. There was primary union of the wound and he was discharged on 7th February 1955. On follow-up on 14th March 1955, he was much healthier and had no complaints.

Case No. 3

T.S., 25 years male, admitted to East Surgical Ward of V.J. Hospital, Amritsar on 21st July 1955 as a case of sub-acute intestinal obstruction. The complaints started 6 months before admission with recurring colicky attacks in abdomen lasting a day. Ten days before admission pain became persistent and located mostly around umbilicus and was accompanied by increased gurgling sounds in the abdomen. It was relieved considerably whenever he passed wind. There was no constipation, vomiting or rise of temperature. Since four days before admission he started getting distended in the central part of abdomen. He had not passed a motion for 2 days before admission, though he was passing wind.

General examination revealed a well built, well nourished individual, slightly anaemic, pulse 54/min., good in volume and tension. Examination of abdomen revealed central distension with visible peristalsis, PR-Faecal matter present in rectum.

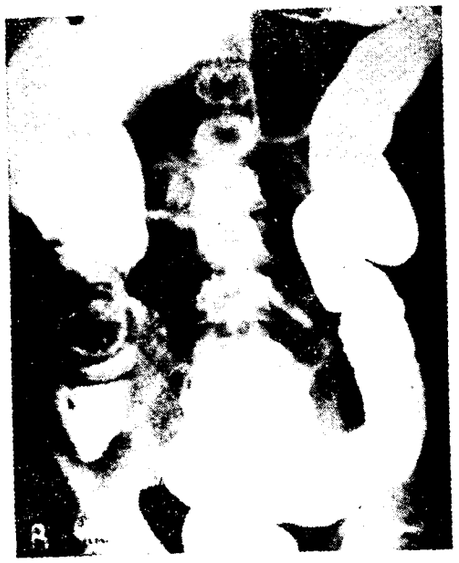

Fig. 5

Case III. Roentgenogram after barium enema, showing filling defect in ascending colon, suggestive of intussusception.

A low enema was administered with good result.

E.S.R. on 22nd July, was 60 mm., first hour Westergren. Urine—no abnormality, X-ray chest—normal. Barium enema examination on 23rd July showed a filling defect in the colon above the caecum-appearances suggestive of intussusception. (Fig. 5).

On 26th July 1955 laparotomy was performed through a right transverse incision. Distended loops of small gut presented in the wound. These were traced to an enteroenteric intussusception, which reached the ileocaecal valve, but no further. It was easily reduced when the apex was found to consist of a very firm growth projecting into the lumen. About 3 feet of small gut bearing the growth was resected and end to end anastomosis done. Laparotomy wound closed in layers with glove drain in subcutaneous tissues.

Pathological report : A piece of small intestine, 5.5 cm. in diameter. At its end there is an encapsulated growth size of 3 × 3 cm. projecting into the lumen. Cut surface of white uniform appearance. Beyond the growth there is a markedly dilated length of small gut, 28 c.m. in diameter. (Fig. 6).

Fig. 6

Same case as in Fig. 5. Excised portion of ileum showing huge dilatation proximal to an intraluminal pedunculated fibroma.

Histology : Fibroma undergoing myxomatous degeneration (Fig. 7).

Patient had a smooth post-operative period with primary union of the wound and was discharged on 5th August 1956.

Fig. 7

Same case as in Figs. 5 and 6. Microphotograph, high power shows fibroma undergoing myxomatous degeneration.

Case No. 4

B.D., 27 years male, was admitted in East Surgical Ward, V.J. Hospital, Amritsar on 6th August 1955 with history of attacks of colicky pain in abdomen, moving ball of wind and constipation of 10 days duration. Two days before admission the severity of pain increased, constipation became absolute and abdomen started distending. He had also repeated vomits. In the past history he described attacks of constipation off and on over the last 2 months.

On examination, general condition fair, tongue dry, pulse 100/min., B.P. 130/90 Hg. Abdomen showed central distension and visible small gut peristalsis. Palpation did not reveal any mass or localised tenderness. P.R.— No abnormality.

Intranasal gastric suction and I.V. 5% Glucose saline drip started. The same evening laparotomy was performed through a left paramedian incision. An ileocolic intussusception going upto lower part of Sigmoid was found. The outer layer was greenish black in colour and while an attempt was being made to reduce, it got ruptured. The apex of the intussusception was gangrenous and bore a dark coloured growth. Resection and end to end anastomosis was performed. 1 gm. of Streptomycin dusted into the wound and the wound closed in layers without drainage. Patient received 650 cc. of blood. I.V. Glucose saline drip and Penicillin and Streptomycin continued post-operatively. Stitches removed on 10th post-operative day. There was primary union of the wound. Patient discharged on 17th August 1955.

Pathological report : A piece of small gut 50 cm. in length with an intussusception 20 cm. long and a hard growth 4 cm. × 2½ cm. × 2 cm. at the apex.

Histological examination : Showed this to be a fibroma with a hyaline change. (Fig. 8).

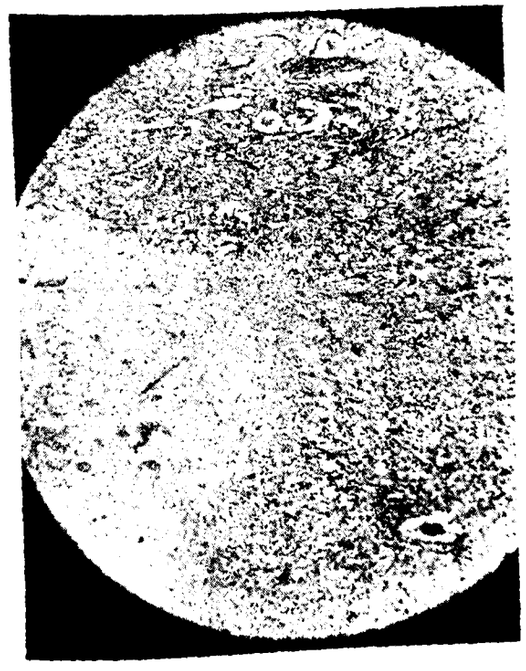

Fig. 8

Case IV. Microphotograph, low power shows fibroma with hyaline change.

Case No. 5

S.K., 28 years female, admitted in East Surgical Ward, V.J. Hospital, Amritsar on 24th January 1957 with acute pain in abdomen starting in right iliac region and then shifting to umbilical region of one day's duration. This was accompanied by vomiting, gurgling sounds, and constipation. General condition good. Tenderness all over abdomen but no distension. Relieved by passage of flatus tube. Serum amylase and urinary diastase showed normal values. There was no recurrence of pain for a week and patient was discharged with advice to report if pain recurred.

She was re-admitted 2½ months later with similar complaints, but there was no constipation. There was tenderness round about the umbilicus but no distension or guarding.

Relieved by injection of morphine sulphate gr. ¼ and atropine sulphas gr. 1/100. I.V. 5% glucose drip and gastric aspiration started. Pain recurred once or twice in 24 hours. Barium meal study of stomach and duodenum did not reveal any abnormality. I.V. Cholecystography showed a normal functioning Gall bladder. Two weeks after admission there was moderate distension of abdomen and a firm, slightly tender, mobile mass was palpable in right iliac fossa. The distension was relieved but an ill-defined mass persisted in hypogastric and right iliac regions. Barium meal study of small gut a week later revealed distended loops of small gut (Fig. 9). Patient was running a high intermittent temperature for which reason laparotomy was postponed till 6th April 1957, when she developed distension with visible peristalsis.

Fig. 9

Case V. Roentgenogram after barium meal shows dilated coils of small gut.

Laparotomy revealed markedly tense sausage shaped distended coil of small gut occupying the pelvis, the gut proximal to which was dilated. This was an enteroenteric intussusception, the apex of which was 1½ feet from the neck and the outer layer showed patches of necrosis at places. Resection with end to end anastomosis was carried out. Wound closed in layers without drainage. Patient made an uneventful recovery and was discharged on 17th April 1957.

Specimen : Intussusception 40 cm. from neck to apex. On opening up it revealed a narrowing at its apex, proximal to which was a pedunculated simple growth, dark red and smooth 7 × 5 × 5 cm. projecting into the lumen. There was firm dark nodule 2 × 1.5 cm. in the mesentary of excised gut (Fig. 10).

Histological report : The growth projecting into the lumen and the nodule in the mesentary showed microscopic structure of a cavernous Haemangioma (Figs. 11 and 12).

Case No. 6

A.D., 29 years female, admitted on 26th November 1957 with history of severe generalized pain in abdomen of 10 days' duration and absolute constipation for 6

Fig. 10
Same case as in Fig. 9. Excised portion of small gut showing an intraluminal pedunculated haemangioma on the left, and a firm dark nodule (cavernous haemangioma) in the mesentary.

Fig. 11
Same case as in Figs. 9 and 10, low power. Normal mucosal pattern in upper part of the field, with structure of cavernous haemangioma in the lower part.

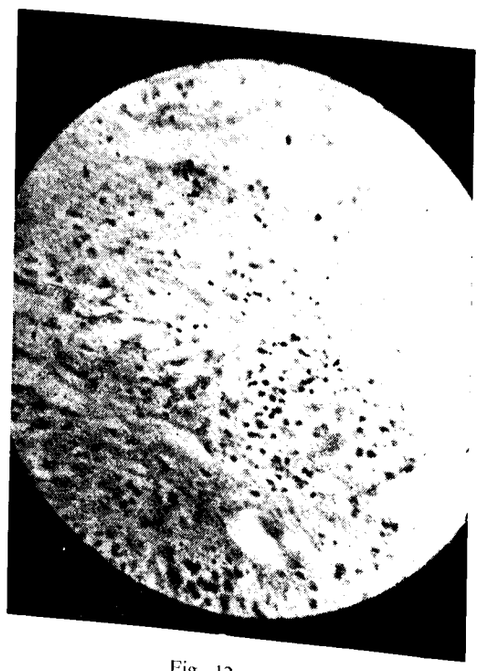

Fig. 12
Same case as above, high power.

Fig. 13
Case VI. Roentgenogram after barium enema reveals a filling defect in the ascending colon.

days. She was conscious of slight yellowness of eyes since 5 days.

On examination, moderately built poorly nourished and somewhat dehydrated young woman, pulse 90/min. B.P. 110/80 Hg. Temp. 100.5 F. Slight jaundice present. Lower part of abdomen distended with prominent coils of small gut. No localised tenderness of mass palpable. P.R. and P.V. examinations did not reveal any abnormality.

Gastric suction and I.V. 5 % glucose saline drip started. Straight skiagram of abdomen showed a moderately distended coil of small gut in lower abdomen with presence of gas in line of colon. Hb. 9 gm.%, T.L.C. 27,000/c.mm., D.L.C. 94/6/0/0. Penicillin and Streptomycin I.M. started. Distension and Temp. persisted though patient was moving her bowels. Barium enema revealed a filling defect in ascending colon, Caecum was not visualized (Fig. 13) Liver function test did not reveal any gross abnormality.

Laparotomy performed on 12th December 1957 through a transverse incision in right iliac fossa, revealed an ileocolic intussusception. The apex had reached middle of transverse colon. There were marked adhesions at the neck making reduction impossible. Right hemicolectomy was performed with end to end anastomosis. Patient had a smooth recovery. Wound healed by primary intention. Discharged on 24th December 1957.

Specimen showed that the intussusception had started by invagination of base of caecum 1.5 cm. outside the ileocaecal valve. This area showed an irregular ulcer with sloping margins, without oedema or undermining. On cutting this area, a dark nodule 1 c.m. in diameter was seen in the wall of caecum (Fig. 14).

Fig. 14
Same case as in Fig. 13. Excised specimen showing a small haemangioma in the wall of the caecum.

Histological examination revealed capillary Haemangioma (Figs. 15 and 16).

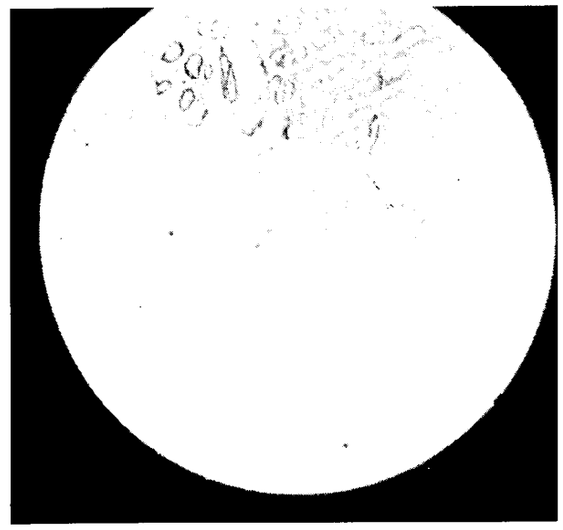

Fig. 15
Same case as in Figs. 13 and 14, low power. Shows the ulcer in the upper part of the field with overhanging edge of mucosa. In the right part of the field is seen the structure of capillary haemangioma.

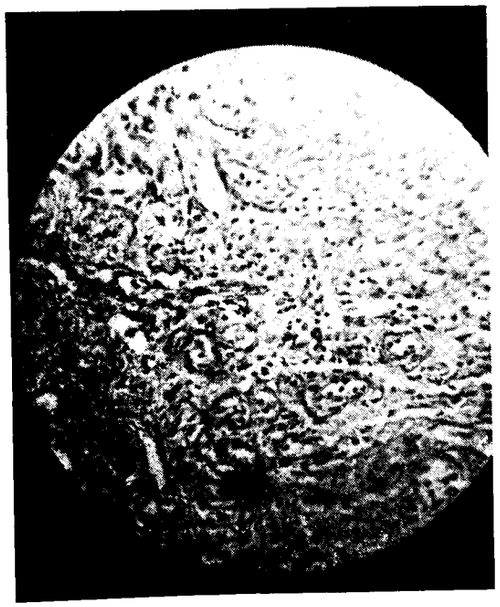

Fig. 16
Same case as above, high power.

Summary

1. Six cases of simple tumours of small bowel and caecum causing intussusception in adults are presented.
2. Recent literature concerning benign neoplasms of small intestine and intussusception in adults is briefly reviewed.
3. It has been emphasized that the possibility of finding a benign neoplasm of small intestine as a causative lesion of patient's symptoms or as an incidental finding at laparotomy should be kept in mind.
4. It has also been stressed that intussusception in adults is not as uncommon as it is usually believed to be and that simple tumours are present in a majority of such intussusceptions.

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PANCREATO-DUODENECTOMY FOR CARCINOMA OF AMPULLA OF VATER

BY

R. S. GUPTA, PATNA

Carcinoma of the ampulla of Vater being a rare condition is considered worth describing. One such case was admitted and treated in the Eastern Railway Hospital at Dinapore. The full case history and operative technique as carried out are described in some detail.

The patient aged 45 years was admitted for jaundice for the past 2 months. He had been having vague epigastric distress for the past one year. The jaundice had been fluctuating in nature with no anorexia or loss of weight or bowel disturbance. On clinical examination, there was tenderness over the right hypochondrium, liver enlarged 2 fingers, and the gall bladder was palpable. Laboratory examination showed the stool clay-coloured. Urine showed bile salts and bile pigments. Icteric index 150 Hb. 11.5 gm. Blood pressure 120/80.

An exploratory laparatomy was carried out. On examination at operation the gall bladder was found distended ; the common bile duct distended considerably to about the size of the thumb. No stone was palpable in the gall bladder or C.B.D. The C.B.D. was opened. A probe could not be passed into the duodenum as there was apparent obstruction at the ampulla. A duodenostomy was done and a growth was felt projecting at the ampullary region. The lymph node by the side of the C.B.D. was enlarged. The C.B.D. was drained by a T-tube and the abdomen was closed. As there was no blood available for transfusion, it was considered wise to operate on him after 3 or 4 days, and also to improve his liver function somewhat by drainage.

The patient was re-operated on two days later with the intention of carrying out a pancreato-duodenal resection. The original Kocher's incision was extended by a right para-median incision. The duodenum was mobilised by incising the peritoneum on its lateral side. The gastro-colic omentum was then opened after mobilising the hepatic flexure. The inferior pancreato-duodenal artery was ligated. The lesser omentum was opened. The right gastric, anterior duodenal and gastro-duodenal arteries were tied. A finger was passed in front of the C.B.D. to emerge in front of the superior mesentric vessels at the inferior border of the pancreas. The stomach was incised 2 inches above the pylorus. The pancreas was then cut at its neck and the distal pancreatic duct was isolated and a catheter passed in it. Bleeding was controlled from the pancreatic surface by applying non-absorbable silk stitches. The C.B.D. was then cut. The ligament of Treitz was then divided and 6 inches of jejunum beyond the duodeno-jejunal junction was mobilised and divided. The proximal end of the jejunum with the fourth part of the duodenum was then delivered to the right from behind the superior mesenteric vessels. The removal of the cut end of the pancreas from its bed as a last stage of the operation presented some difficulty, but the vessels were carefully sought and ligated. The whole specimen was removed en block. Some enlarged lymph nodes were found along the superior and inferior borders of the pancreas.

This having been done, an end-to-end gastro-jejunostomy, and end-to-side pancreato-jejunostomy, and side-to-side cholecysto-jejunostomy was carried out. An entero-enterostomy was done just distal to the gastro-jejunostomy.

End-to-end Gastro-Jejunostomy :—About 3 inches of stomach, proximal to the pylorus, was removed. Part of the stomach was closed and to the remaining, end-to-end gastro-jejunostomy carried out.

Pancreato-Jejunostomy:—The pancreatic duct was found distended after removing the head of the pancreas. A mucosa to mucosa anastomosis was done between the pancreatic duct and the jejunum over a polythene tube. The anastomosis was covered by bringing the pancreatic tissue to the jejunum by non-absorbable sutures, both anteriorly and posteriorly.

Difficulty was experienced in anastomosing the common bile duct to the jejunum because of the T. tube, which was inserted in the C.B.D. at the previous operation, and therefore, it could not be mobilised effectively. The end of the C.B.D. was closed and a side-to-side cholecysto-jejunostomy was done. The T. tube was left in the C.B.D. distal to the anastomosis. Then an entero-enterostomy was done distal to the gastro-jejunostomy so as to by-pass

the stomach contents. The abdomen was closed with a drain at the anastomotic site.

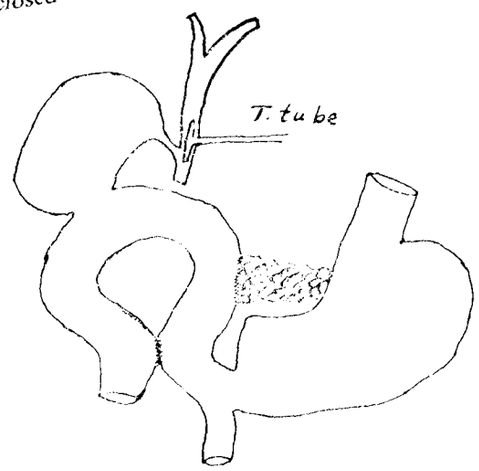

Fig. 1
Appearance at completion of operation.

Fig. 2
Specimen as seen after removal showing the lower part of stomach, duodenum, head of the pancreas and upper part of the jejunum.

Both the procedures were done under light spinal anaesthesia giving 17 c.c. of liq. nupercain on each occasion. Total operative time taken for the second operation was $4\frac{1}{2}$ hours, and the spinal anaesthesia had to be supplemented by gas oxygen aether after 3 hours of operating time. Intravenous Pethedine, 100 mg. total, was given during the operation in 30 mg. doses at a time. 600 c.c. of blood, 2 bottles of Plasmosan and 2 bottles of 5% glucose was given during the operation. The blood pressure remained in the region of 100-110 throughout, and the pulse between 90 and 100 per minute throughout the operation.

The post-operative course was uneventful. The T. tube drained bile for one week gradually lessening in amount. The drain was removed on the 4th day. The bile started appearing in the stools after one week. The T. tube was gradually blocked till it was kept closed for full 24 hours after the 20th day of operation. A choledochogram showed the lipiodol in the intestines via the cholecysto-jejunal anastomosis, and the T. tube was removed.

The histopathological report was as under :

"The sections from the growth in the duodenum shows acinus forming epithelial neoplasm. The cells are large with very big vesicular prominent nuclei. The acini are of varying shape and size. Papillomatous projection of neoplastic cells are also seen projecting into the acini. The Anaplastic cells have also infiltrated into the muscle coat. Chronic inflammatory cells are found scattered in the superficial part of the neoplasm. Histological diagnosis — Adeno-carcinoma.

Fig. 3
Microphotograph of the specimen.

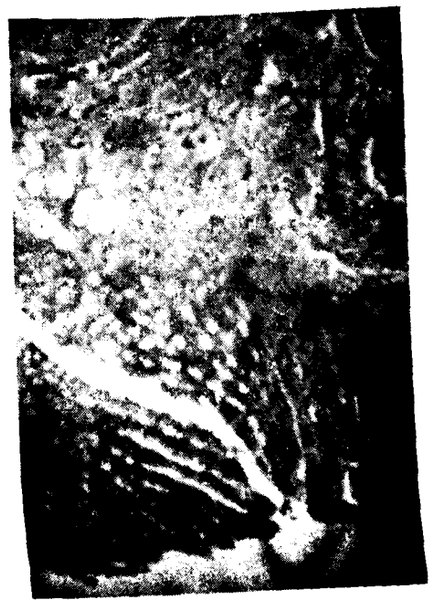

Fig. 4
Microphotograph of the specimen.

Follow-up :—The patient is well 3 months after operation with no complaint and is doing his normal duties.

Summary

A case of pancreato-duodenectomy due to adeno-carcinoma of the ampulla of Vater is presented. The operative technique used is given in detail. It is suggested that when the cystic duct enters the C.B.D. at a higher level, a cholecysto-jenunostomy, rather than a choledocho-jejunostomy will give more satisfactory results.

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A STUDY OF BONE MARROW AND PERIPHERAL BLOOD IN BREAST CARCINOMA

BY

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The present study of bone marrow and of peripheral blood in Breast Carcinoma was undertaken with a view to assess their clinical value in the diagnosis, progress and therapy in different stages of the disease.

This work was aimed at finding the following facts :

1. Difference in the picture in breast carcinoma as compared with those in the control series of non-malignant cases admitted during the same period.
2. Changes in the sternal bone marrow in different stages of malignant disease of the breast.
3. The effects of irradiation therapy on bone marrow and peripheral blood.
4. To detect malignant cells and other abnormal cells in bone marrow and to compare the results with the radiological findings.

In the present investigations 60 bone marrow and complete peripheral blood examinations were conducted on 50 female adults admitted to the Department of Surgery, Lady Hardinge Medical College and Hospital, during the period of one year from September, 1956 to August, 1957.

This included 25 consecutive cases of breast carcinoma diagnosed clinically, and a control series of 25 patients of the same age group, *i.e.*, between 20 to 60 years admitted for lesions other than breast carcinoma.

The cases of breast carcinoma were considered in the following four groups :

Group A	Cases
Operable cases (*i.e., in stages I & II*) who had Radical Mastectomy followed by post-operative irradiation therapy	6
Group B	
Inoperable who had only Deep X-ray therapy as a palliative measure ...	6
Group C	
Follow-up cases (operated on in the previous years and had irradiation) admitted for check up during this period	7
Group D	
Too advanced for operative or irradiation therapy and had only hormone treatment	6
Total ...	25

Incidentally about the same number of patients were seen in each group.

Group A and B consisted of 12 patients on whom bone marrow studies were done pre-operatively and also in 8 cases following the course of Deep X-ray therapy post-operatively. In two cases the investigations were repeated again after an interval of 4 months.

Method : Using standard sternal marrow needles (Salah's) under local anaesthesia with 1% novocaine for infiltration of the soft tissue, aspiration of sternal marrow was done. On an average ½ to 1 c.c. of the marrow was aspirated except in those who already had irradiation therapy – in the latter it was not always possible to obtain this amount. A similar procedure was carried out in the control cases.

In each case four direct smears were made. In the cases of breast carcinoma the remaining aspirate was transferred to Wintrobes oxalate tubes and two additional slides were prepared after concentration for detection of malignant cells. The slides were stained with haematoxylin and eosin after fixation with 95% alcohol. For the differential 500 cells were examined.

In addition complete peripheral blood examination was done in each case. This included Total WBC and RBC, Differential count, Hb. in gms. (Sahli's method) and erythrocyte sedimentation rate (Wintrobe's method).

Findings

In 25 control cases differential count of the sternal marrow showed the following mean average values :

Table I

Myeloid Cells		Premature Erythrocyte	
Polymorphs	45.50	Promegalbolast	0.71
Lymphocyte	13.66	Normoblast	17.78
Eosinophils	2.40		
Monocyte	0.84		
Basophils	0.20		
Premmyelocyte	0.82		
Myelocyte Neutrophils ...	3.12		
Myelocyte Eosiniphils ...	0.32		
Metamyelocyte Neutrophil ...	13.00		
Metamyelocyte Eosinophil ...	1.50		
	81.51		18.49
			= 100%

Mycloid/Erythroid ratio : 3.75/1

In 25 cases of breast carcinoma classified in 4 groups the following were the findings :

In Group A—6 cases : 5 patients had Radical Mastectomy and a full course of deep X-ray therapy, *i.e.*, 30 exposures 4000 r. to 6000 r. units. One case No. 6 had simple mastectomy with excision of axillary glands on account of her general condition. She was 6 months pregnant and was more advanced than the other patients in this group.

In these cases bone marrow and peripheral blood studies were done both in the pre-operative and in the post-irradiation periods, to study the effects of irradiation treatment as shown in Table II.

Case No. 3 is of particular interest. She had Radical Mastectomy on the left side for stage II growth and within 2 weeks of operation during the course of post-operative deep X-ray therapy, she developed a mass in the right breast along with mobile glands in the right axilla. Simple Mastectomy with excision of glands was done on the right side after completion of 30 deep X-ray exposures. The second operation was followed by a second course of irradiation. Sternal marrow smears were not possible after the last course or irradiation due to extensive skin infiltration involving the entire circumference of the chest wall, but the bone marrow smears were examined after the first course of irradiation. Certain uncommon cells were noted, these were larger than the other premature cells with eccentric hyperchromatic nuclei. Cytoplasm was basophilic and clear, these were probably abnormal normoblasts appearing in response to irradiation.

Group B is comprised of 6 patients who were inoperable and had only palliative deep X-ray therapy. Bone marrow and peripheral blood studies were carried out on admission and repeated after the deep X-ray 4000 r.–6000 r. units. Details of findings are shown in Table III.

TABLE II—Showing sternal Bone Marrow and Peripheral Blood findings in Group A cases

Serial No.	Age	Period of investigation	Bone Marrow: Poly	Lymph	Eos	Mono	Baso	Prem	Myel Neut	Myel Eos	Meta Neut	Erythrocyte: Pro. Ery.	Early Norm	Int. Norm	Late Norm	M/E ratio	ESR mm/hr.	Hb. Gm.	Peripheral Blood: RBC Mil.	WBC cmm.	Poly	Lymph	Eos	Mono	Remarks
			PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT						PER CENT	PER CENT	PER CENT	PER CENT	
1	42	(*a*) Pre-operative ...	51.7	10.75	2.5	4.0	...	0.75	3.5	0.5	10.75	2.0	5.25	5.25	4.0	4.33/1	39	11	...	5000	68	32	...	...	
		(*b*) Immediately after irradiation..	70.0	18.80	2.0	2.0	...	...	0.5	...	1.5	0.5	1.5	4.0	...	12.2/1	24	10	3.8	5800	84	8	6	2	
		(*c*) 4 months' post-irradiation.	57.2	32.00	1.0	...	0.6	...	1.4	...	3.6	0.2	...	1.0	3.0	15.1	20	13	4.16	6800	67	27	3	3	
2	40	(*a*) Pre-operative ...	40.5	15.75	6.25	2.5	0.5	1.75	3.25	...	9.0	1.0	3.50	7.25	8.25	3.17/1	44	12	...	9200	62	34	2	2	
		(*b*) Immediately after irradiation.	63.5	18.5	16.5	1.5	No			premature			cells			seen	27	12	4.24	6600	85	7	7	1	
3	55	(*a*) Pre-operative ...	45.75	15.0	2.5	0.5	1.25	0.5	3.5	...	9.5	1.0	3.0	10.5	7.0	3.0/1	24	13	4.56	5600	74	15	10	1	Cells resembling myeloid cells observed.
		(*b*) Immediately after irradiation.	56.5	27.0	1.0	1.5	...	0.75	1.25	...	3.0	0.25	0.25	6.5	2.0	7.5/1	26	12	4.35	5000	81	8	5	4	
4	35	(*a*) Pre-operative ...	43.0	18.4	1.0	...	...	...	2.2	...	18.2	0.2	0.6	4.0	12.0	3.8/1	30	13	4.54	5600	58	34	4	4	These cases are being treated by deep X-ray post-operatively.
		(*b*) Immediately after irradiation.	58.4	21.2	4.0	1.8	0.6	...	1.2	...	4.0	...	0.6	2.2	4.2	10/1	20	13.5	3.64	4700	72	28	...	...	
5	40	Pre-operative ...	41.4	17.6	2.6	...	...	0.6	1.0	...	14.0	1.0	0.8	4.4	14.8	3/1	20	13	3.84	6600	48	40	8	4	
6	30	Pre-operative ...	49.4	14.0	3.2	...	...	0.6	1.2	...	18.0	...	0.4	3.2	8.8	5.9/1	48	10	3.41	6800	44	55	...	1	

Table III—Showing sternal Bone Marrow and Peripheral Blood findings in Group B cases

Serial No.	Age	Period of investigation	Myeloid Cells: Poly	Lymph	Eos	Mono	Baso	Prem-	Myel Neut	Myel Eos	Meta Neut	Erythrocyte (Premature): Myelo Ery.	Pro. Ery.	Early Norm	Int. Norm	Late Norm	M/E ratio	ESR mm/hr.	RBC Mil.	Hb. Gm.	Peripheral Blood: WBC cmm.	Poly	Lymph	Eos	Mono	Remarks
			PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT						PER CENT	PER CENT	PER CENT	PER CENT	
7	30	(*a*) Pre-irradiation ...	44.0	12.75	1.5	5.75	1.0	1.0	2.25	0.25	11.75	...	0.5	3.50	7.50	8.00	3.29/1	46	...	11	5000	67	30	3	...	Malignant cells present in the marrows mears Markedly hypocellular marrow.
		(*b*) Immediately after irradiation.	69.0	8.0	14.5	...	...	1.0	1.0	...	3.25	...	0.25	1.00	1.50	0.50	27.3/1	45	4.4	14	5800	75	24	...	1	
8	55	(*a*) Pre-irradiation ...	53.75	8.50	17.5	1.5	0.25	1.25	2.75	0.75	6.75	...	1.0	4.25	7.75	8.25	3.21/1	18	3.88	10.5	5400	63	30	2	5	Hypocellular marrow.
		(*b*) 20 days after irradiation (30 exposures).	57.50	19.50	5.50	1.5	1.0	...	2.5	...	6.00	...	...	...	3.00	2.50	13.3/1	18	3.74	12.5	6300	72	23	3	2	Hypocellularity increased.
9	49	(*a*) Pre-irradiation ...	54.0	16.75	2.0	0.5	0.75	0.5	2.0	...	5.75	0.5	1.0	1.25	8.25	6.50	3.3/1	34	3.72	12.5	8200	68	27	...	5	Marrow hypocellular.
		(*b*) Immediately after irradiation (38 exposures).	83.0	12.50	2.0	...	...	...	...	...	0.50	...	...	...	2.00	...	42/1	45	4.70	12.5	5200	84	14	2	...	Hypocellularity increased.
10	60	(*a*) Pre-irradiation ...	46.0	19.0	0.40	1.0	...	...	3.8	...	12.80	0.5	0.40	1.80	5.80	8.60	3.8/1	16	4.42	13.0	6300	64	32	3	1	
		(*b*) Immediately after irradiation (27 exposures).	76.50	13.0	0.50	1.5	...	...	...	...	6.00	...	...	...	2.00	1.50	20/1	43	3.42	11.5	5100	83	11	2	4	Hypocellular marrow.
		(*c*) 5 months after irradiation.	53.40	27.80	2.0	1.2	0.2	...	4.4	...	9.20	...	0.20	0.20	1.80	3.60	11/1	40	3.81	12.5	4400	69	29	1	1	Marrow hypocellular.
11	30	Pre-irradiation ...	47.80	17.80	2.4	0.6	...	...	2.4	...	14.80	0.5	0.40	0.80	4.60	7.60	5/1	27	4.72	10	7000	78	19	2	...	Malignant cells present.
12	60	Pre-irradiation ...	42.80	26.60	3.8	0.2	...	0.6	1.8	0.2	11.00	...	...	1.00	2.60	8.20	5.2/1	44	3.73	10.25	8000	54	31	13	2	

In this series two patients No. 7 and No. 11 showed malignant cells by concentration method prior to irradiation but not after the therapy. The cytoplasm of these cells was approximately 3 times that of the premyelocyte cells with ill defined outline, the cytoplasm and nuclei ratio was thus altered. The nuclei were multiple and hyperchromatic and mitotic figures were present.

Group C : Consisted of follow-up cases 7 in number. These patients had operative and irradiation therapy from 6 months to 3 years previous to re-admission to the hospital during this period of study for check up only ; the findings are shown in Table IV. There was general hypocellularity but the myeloid/erythrocyte ratio was the same as in Group A and B before irradiation. In the case No. 18 malignant cells resembling those seen in cases Nos. 7 and 11 were detected in the bone marrow.

Group D : Consisted of 6 cases who were too advanced either for operative or deep X-ray therapy and 4 of them were treated palliatively with Hormone–oestrogen therapy. Details are shown in Table V. No malignant cells were found but hypocellularity of the bone marrow was marked. In the case No. 20 it was not possible to obtain any aspirate and only small amounts were aspirated in the other cases in this series. Except in case No. 23 myeloid erythrocyte ratio remained within normal limits in this patient it was 10/1 due to increase of polymorphs and relatively low immature erythrocytes.

It was noted that immediately following a course of irradiation therapy there was a rise of polymorphs in the bone marrow with a relative fall in the premature myeloid and erythrocyte cells. This corresponded to the rise in polymorphs in the peripheral blood although the total white cell count was not

TABLE IV *Showing sternal marrow and Peripheral Blood findings in Group C cases*

Serial No.	Age	Duration after irradiation.	Bone Marrow Myeloid Cells								Erythrocyte (Premature)				Peripheral Blood								Remarks.
			Poly	Lymph	Eos	Mono	Baso	Premy	Meyl Neut	Meyl Eos	Meta Neut	Pro. Ery.	Early Norm	Int. Norm	Late Norm	M/E ratio	ESR mm/hr.	Hb. Gm.	RBC Mil.	WBC cmm.	Poly	Lymph	Eos	Mono	
			PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT			PER CENT			PER CENT	PER CENT	PER CENT	PER CENT	
13	36	.....	42.75	13.50	4.50	...	1.00	0.50	1.25	...	4.75	0.25	3'75	13.50	13'25	2.17/1	38	12.5	3.75	12,000	91	7	...	2	Interrupted course of irradiation 20 days.
14	35	6 months ...	55.60	14.00	1.20	0.80	...	0.40	3.00	...	6.40	0.80	1.40	8.20	9.00	3/1	38	9.5	3.19	10,300	90	9	1	...	Secondaries in the lungs.
15	55	6 months ...	60.00	20.00	1.40	1.00	...	...	0.80	...	8.00	...	0.20	5.00	3.00	8.4/1	36	11.5	4.23	5,000	70	26	4	...	
16	40	1½ years ...	52.00	16.00	1.25	0.75	0.25	1.75	5.25	...	9.00	1.00	1.50	5.50	5.50	5.16/1	48	12	...	6,200	65	33	2	2	
17	44	1 year and 8 months ...	61.25	15.00	2.50	1.75	0.50	1.50	3.00	...	5.00	0.75	1.00	4.75	3.00	7.97/1	24	14	4.35	9,500	74	25	...	1	
18	50	2 years ...	40.25	19.25	6.75	1.25	...	0.25	3.50	...	4.75	1.00	2.00	9.00	14.25	2.19/1	50	10	3.75	7,000	70	26	4	1	Malignant cells present. Secondaries in the sternum.
19	52	3 years ...	31.00	32.00	1.00	...	...	2.00	3.50	...	6.00	0.50	4.50	12.00	7.50	1.74/1	40	12	...	6,400	54	44	...	2	

TABLE V—Showing the Bone Marrow and Peripheral Blood findings in Group D cases

Serial number	Age	Bone Marrow Meyloid Cells									Erythrocyte (Premature)				Peripheral Blood								Remarks
		Poly	Lymph	Eos	Mono	Baso	Premy	Myel Neut	Myle Eos	Meta Neut	Pro. Ery.	Early Norm	Int. Norm	Late Norm	M/E ratio	ESR mm/hr.	Hb. Gm.	RBC Mil	WBC cmm.	Poly	Lymph	Eos	Mono	
		PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT	PER CENT						PER CENT	PER CENT	PER CENT	PER CENT	
20	30	No Bone Marrow Aspirated									(Dry Puncture)												
21	55	45.50	15.50	2.75	0.50	0.25	1.25	2.00	0.50	6.25	1.00	2.00	12.25	8.25	2.6/1	21	12.5	5.00	7,000	70	30	...	...	
22	50	43.00	16.00	1.75	0.75	...	1.75	3.00	...	10.25	1.25	2.50	10.50	9.25	2.5/1	...	12.25	...	8,000	56	40	4	...	
23	30	68.25	12.00	1.25	2.00	...	0.50	2.25	...	6.50	...	0.75	4.25	2.25	10/1	51	10.50	3.37	9,400	71	26	2	1	
24	42	38.40	14.40	0.60	...	...	0.40	3.60	...	14.00	1.00	1.20	10.60	15.00	2/1	28	11.50	3.73	7,000	70	29	1	...	
25	25	34.25	17.00	1.50	1.50	...	0.50	5.25	0.25	2.00	1.25	6.75	14.75	14.75	1.17/1	24	5.50	2.71	8,000	80	16	1	3.00	

much altered. On the whole the marrow showed hypocellularity and ESR was lowered following irradiation therapy.

Control series

The control series consisted of 25 cases who were admitted to the hospital for diseases other than malignant lesions during the same period. In all the cases Haemoglobin was above 10 gms. The following were the lesions : 5 cases admitted for fibroadenoma breast, 2 for umbilical hernia, 4 for Benign tumours of the neck and limbs, 1 for ureteric calculus, 1 for burn contracture, 1 for hydatid cyst, 9 for Tuberculosis in the abdomen and axillary glands and 2 for chronic osteomyelitis.

Discussion

In the recent years because of the hazards of the nuclear weapons and the discovery of radioactive substances greater interest is being taken in the study of effects of irradiation on the Haemopoietic tissues particularly the blood and bone marrow. Several workers have reported the increasing incidence of Leukaemia as a result of irradiation. The scope of our work has, however, been limited to the study of bone marrow and peripheral blood in mammary carcinoma in different stages of the disease, and to find the effects of irradiation on the marrow, as this method of treatment is now extensively used for such patients.

Ackermann (1956) has contributed valuable observations from the study of bone marrow aspirates from the vertebrae of patients with breast carcinoma since metastases occur very frequently in these cases. However, other workers Kreyberg and Poppe (1940) and Kingsley Pillar and John Marks (1956), have found sternal bone marrow just as useful for the purpose of diagnosis. The sternal marrow aspirate is easily obtainable with a simple technique from the sternum as this is a subcutaneous bone and has only a thin cortex and the marrow space is easily reached with a marrow needle. This method has been in use for a long time for diagnosis of blood and bone marrow diseases such as anaemia, leukaemia, etc.

In the present study of comparison of the bone marrow from 25 cases of mammary carcinoma with that from 25 non-malignant cases used as controls reveals certain interesting features. On the whole the marrow from the malignant cases showed hypocellularity, particularly affecting the myeloid series of cells. The myeloid erythroid ratio remained within normal range.

The peripheral blood picture showed no significant changes except that the polymorphs were slightly raised from 63 to 69% in the differential count although the total WBC remained constant. Haemoglobin and the total red cell count also remained unaltered except in advanced stages with skeletal metastases as in case No. 25 with Hb. of only 5.5 gms. and total RBC only 2,500,000 c.mm. It is possible that the constant level was maintained (in the majority) as a result of anti-anaemic treatment with iron, vitamins and blood transfusion in the pre and post-operative periods. An interesting feature was the high erythrocyte sedimentation rate noticed in all the malignant cases steadily rising with the advancing stage of the disease prior to treatment ; following operative and irradiation therapy as in Group A, B and C there was a gradual fall in ESR. This of course is also found in other non-malignant lesions mainly infectious ; the ESR rises during the activity of the disease and falls with the control of the disease. Therefore, it is not a characteristic feature of malignancy but it is an useful indicator in the control of these cases during the deep X-ray therapy.

In addition, certain observations were made on the bone marrow following irradiation therapy which usually consisted of 30 exposures, the total dosage being 4000 r. to 6000 r. on an average. In certain cases in response to irradiation abnormal cells appeared in the bone marrow, some resembled plasma cells while others showed basophilic cytoplasm with eccenteric nuclei resembling Myeloma cells. Although 19 out of 25 patients had deep X-ray therapy there was no suggestion of leukaemic changes. Jainet and Amy (1956) have described " secondary cell patterns " in bone marrow of malignant cases, but our series is not large enough for such a conclusion. On further careful examination of the bone marrow smears prepared after concentration of the aspirate it was possible to detect malignant cells with the characteristic features similar to those described by other workers in 3 out of 25 malignant patients, *i.e.*, in cases Nos. 7, 11 and 18. While in case No. 24 suspicious cells were noticed but the smears were not included in the positive group. These cells had characteristic features, they were large although not all found in groups or clumps, the cytoplasm was basophilic, homogeneous matrix, outline not very clear and the nuclei were hyperchromatic showing mitotic forms. In case Nos. 7 and 11 there was

no clinical or radiological evidence of distant metastases though in No. 18 X-ray of the spine was suggestive of skeletal metastases.

Although the present series is not large it does compare favourably with the findings of the other workers in the western countries. Recently Kingsley Pillers and John Marks (1956) published their results of sternal bone marrows into positive, suspicious and negative groups according to the presence or absence of malignant cells, other abnormal cells, and changes in the bone marrow. Their percentage of positive smears was 6.2% in the total series of cases and out of these 62% were positive in those patients who had no radiological evidence of the disease. It is interesting to note that in our series of breast carcinoma the incidence of positive smears was higher, in 3 out of 25 cases, *i.e.*, 12% in the total number, and in 2 of these cases or in 66.6% positive bone marrows were found in the patients with no evidence of metastases radiologically. It is doubtful that the higher incidence in the present series was due to the fact that only cases of breast carcinoma were included, and because of the proximity of the primary tumour to the sternum the latter was affected by direct spread. In our experience the skeletal metastases from mammary carcinoma occur most frequently in the spine and in the extremities chiefly in the femora so the only possibility of spread in these bones is via the blood. Moreover, if the sternum is affected directly or via the lymphatics it would be the bony structure that would be more likely affected rather than the marrow and the X-ray findings would be positive. But this can now be ruled out by the fact that even in the absence of bony changes and radiographic evidence the sternal marrow was positive for malignant cells. Therefore, early diagnosis of malignant changes in bone marrow has significant clinical value in the progress and in therapy especially in the absence of radiological evidence of metastases. It is not uncommon to find instances of malignant cases who are considered to be in the early operable stage with no positive X-ray findings, and who return within a short period of a successful operation with subsequent metastases. Bone marrow examination may prove useful in detecting the spread of growth in such cases.

Willis (1934) had observed that the blood borne metastases in bones occur initially in the bone marrow and the actual bony changes follow in response to the presence of irritating factors and that the structural changes are effected by osteoblasts and osteoclasts. Shackman and Harrison (1948) proved the above view by investigating the skeletons at autopsies of those patients who had died of malignant disease with distant metastases. They selected those specimens which showed naked eye deposits without apparent bony changes and later confirmed microscopically and took contact radiographs of the excised bones; in more than half the cases X-ray findings were negative. Therefore, they concluded that radiographs do not always exclude the presence of even extensive bone metastases. Later Ardan (1951) confirmed Shackman and Harrison's findings by observing that even when slices of bones are removed the defect in the bone may not be evident radiologically until about 1 c.m. of bony thickness is removed. Therefore, routine radiography does not exclude the presence of bone destruction in malignancy unless different views or tomagraphs are taken. This is important clinically since the diagnosis of metastases is mainly based on routine radiography.

Recently Jainet and Amy (1956) in their very extensive study of bone marrow aspirates from a large series of 4001 patients, found total positive aspirates for carcinoma in 55.5% and diagnosed 18.5% occult cases of malignant growths by this method alone in the absence of any specific clinical evidence of the disease. They were also able to detect the site and nature of the primary growth from the appearance of the malignant cells in marrow biopsy. Kreyberg and Poppe (1940) had also demonstrated similar findings from the study of sternal bone marrow biopsies.

The purpose of the present study is not so much to diagnose the primary growths which in the case of breast is diagnosed clinically in the majority of patients, as to detect metastases or any changes in bone marrow before these become evident clinically or radiologically. This is important from the point of view of progress and treatment of the patients with mammary carcinoma. Operability of the case depends on the extent of the growth, with distant metastases the case is no longer considered operable even though the case may clinically appear to be in the early stage. Therefore, examination of the bone marrow has a place in the investigation of such cases.

Conclusion : In view of the positive findings in 12.0% of the total number of cases investigated and with 66.6% showing the presence of malignant cells without radiological evidence, examination of sternal bone narrow is of

definite value clinically. This is particularly important for the diagnosis of metastases and in deciding the prognosis and operability of malignant lesions of the breast.

Summary

1. Sternal bone marrow aspirates and peripheral blood from 25 cases of breast carcinoma and from 25 non-malignant patients as control series were investigated. Total number of 60 sternal punctures were performed in 50 female adults in the pre and post-operative and post-irradiation periods.
2. The cases of breast carcinoma were classified into 4 groups, according to the stages of the growth and the treatment given.
3. Positive smears *i.e.*, malignant cells in the sternal bone marrow were found in 12% of the carcinoma cases. In 66.6% of these there was no radiological evidence of the disease.
4. Certain other changes in the marrow and peripheral blood are described particularly changes in response to irradiation.
5. The literature on the subject is briefly reviewed.

Acknowledgment

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EOSINOPHILIC GRANULOMA OF FEMUR

BY

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In 1940 Otani[12] and Ehrlich and Lichtenstein[10] and Jaffe independently described a destructive lesion of bone consisting chiefly of histiocytes and eosinophiles. The latter authors named the process "Eosinophilic granuloma of bone" and suggested virus as the probable aetiological agent in view of the negative bacteriological cultures. Schairer[13] and Otani[12] and Ehrlich favour trauma as the initiating factor, for not only is a history of trauma usually volunteered by these patients, but the lesion is mostly confined between the ages of 5 to 14, when trauma is extremely common. Gross[8], Farber[6], Green[7] and Farber and Mallory[11] pointed out the identity of this lesion with reticulo-endotheliosis and drew attention to its resemblance to Hand-Schuller-Christian disease, or Xanthoma of bone, or Letterer-Siwe's disease.

Skull, pelvis, ribs, vertebrae, femur and humerus are the sites of predilection. In most of the cases the lesion is solitary but reports of multiple bone involvement are not infrequent. More interesting are the reports of pulmonary involvement accompanying cases of eosinophilic granuloma of bone recorded by Engelberth[5] Holm Telium and Christensen, Arnold[1], Childers[3], Middleton and Schneider and Currens[4] and Popp. Up to date about 200 cases of eosinophilic granuloma of bone have been reported. Because of the benign nature of the lesion and the diaphysial involvement of the long bones, sharing in common clinical and radiological appearances with other inflammatory or neoplastic lesions of the bones, it is very necessary to draw the attention of the clinicians to the not infrequent occurrence of this condition. Since biopsy alone can disclose the real nature of the lesion and to stress the utmost necessity for the same before treatment is undertaken, clinico-pathological findings in a case under our care are recorded.

Case Report

K.M., Hindu boy aged 7 years was admitted under G.E., in Government General Hospital, Guntur, in September 1957 for a painful swelling at the middle of the right thigh of three months' duration. The swelling was noticed to increase slowly in size. There was no history of injury to the site or of fever and none in the family had similar trouble.

Fig. 1

Photograph shows fusiform swelling in the rt. mid-thigh.

Clinical Findings

The boy is moderately well built and active. A fusiform swelling at the right mid-thigh was noticed. (Fig. 1). The skin over the swelling was healthy. The swelling was warm and tender. The soft tissues over the swelling were not adherent. Movements of the joints were not restricted. Regional lymph nodes were not enlarged. Other systems nil remarkable.

Laboratory and other data

Urine	:	Nil. remarkable.
Faeces	:	Round worm ova detected.
W.B.C. count	:	10,200 cmm. P68, L23, E6, and M3.
Serum—		
Calcium	:	9.5 mgms. per cent.
Phosphorus	:	4.5 mgms. per cent.
Alk. phosphatase	:	20.5 K.A. units.
Cholesterol	:	199 mgms. per cent.
V.D.R.L.	:	Negative.

* From the departments of Pathology and Orthopaedics, Guntur Medical College, Guntur, India.

X-Ray

Fusiform swelling of the diaphysis of the right femur is seen. Medulla is enlarged with destruction of the cortex and marked periosteal reaction. (Fig. 2). Based on this, provisional clinical diagnosis of chronic osteomyelitis or Ewing's tumour was made.

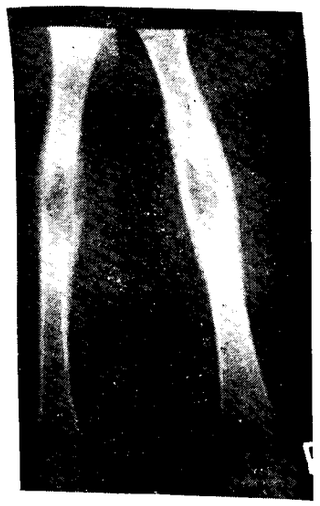

Fig. 2

Reduction X-ray photograph of rt. femur illustrates circumscribed area of destruction of cortex and expansion of medulla—typical of eosinophilic granuloma.

Biopsy Report : (1963/57)

On exploration the cortex of the bone was found to be thickened. A small bit measuring 0.5 cm. was removed from the site of the lesion. The tissue was greyish and soft. Microscopic examination revealed cosinopile cell collections, histiocytes and young fibrous tissue. In places, the histiocytic collections appear to replace the eosinphiles. (Figs. 3, 4, 5, 6 and 7). Usually at the periphery of the lesion commencing fibrosis was noticed. In the nidus of the lesion mostly eosinophiles were observed. The histiocytes presented faintly eosinophilic cytoplasm with occasional fine vacuolation but no inclusions. The lesion was poorly vascular and bore no relation to blood vessels. Giant cells were not noted.

Diagnosis of " Eosinophilic granuloma of the bone " was made.

Treatment

At the suggestion of Dr. S. Venkateswarulu, Radiologist, the patient was given the benefit of deep X-ray Therapy of 3,400 r. spreading over 9 days (from 7th October to 17th October 1957.) The patient was able to use his limb better, tenderness and pain over the right thigh were considerably less. X-ray of the lesion at the conclusion of therapy showed pronounced concentric sclerosis around the lesion but for a small area of translucency yet to be filled up. (Fig. 8). The patient has been advised to visit the clinic periodically for follow-up.

Fig. 3

Photomicrograph illustrates commencing histiocytic reaction around eosinphile aggregates. (H. & E. X270)

Fig. 4

Photomicrograph illustrates preponderant eosinophi cell collections and commencing fibrosis (H. & E. X270)

Fig. 5

Photomicrograph illustrates mixture of eosinophile and histiocytic cell collections composing the lesion (H. & E. X400)

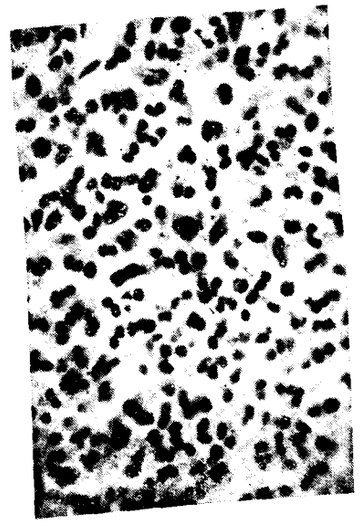

Fig. 6

Photomicrograph illustrates preponderance of binucleate cells characteristic of eosinophiles (H. & E. X400)

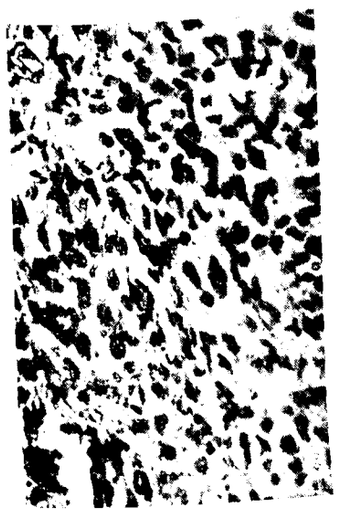

Fig. 7

Photomicrograph illustrates exclusively histiocytic reaction observed in one field. (H. & E. X300)

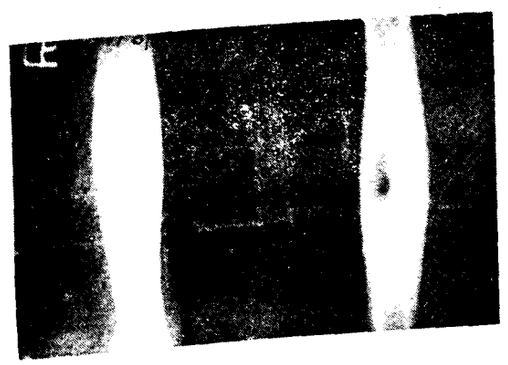

Fig. 8

X-ray reduction photograph (anterior and lateral) of the lesion after deep X-ray therapy shows sclerosis and almost complete repair of the lesion.

Comment

The clinical and microscopic findings of the lesion observed in our case are typical of eosinophilic granuloma of bone. This case emphasises the fact that the diagnosis of this condition is the prerogative of the pathologist and that the implications of this diagnosis in terms of treatment and prognosis demands constant awareness of this lesion in the microscopic examination of certain bone lesions. The pathologist is likely to encounter under the microscope eosinophiles exclusively or mixture of eosinophiles and histiocytes as was noticed in our case or only histiocytes and fibroblasts, all depending on the stage of the lesion at the time of biopsy examination. This has led to different interpretations by the pathologists. Till more is known of the exact significance of the eosinophiles noticed in the bone lesions and their close association with histiocytes, one is sceptical to accept the view that it is one phase of systemic reticulo-endotheliosis. Although Baas[2] reported 4 to 11 per cent blood eosinophilia in 7 of his cases it is hasty to suggest the entire phenomenon to be one of an infiltrative process. Some have recorded total blood fat significantly elevated in 50 per cent of the cases but our case showed normal cholesterol level and this needs further study. What exactly initiates eosinophilic reaction in the bone and sometimes accompanied by pulmonary or other visceral changes is yet to be determined. Experimental and clinico-pathological study of the lesion at different stages may throw further light.

Different modes of treatment have been suggested and the same being based on the experience of each surgeon. Hechberg and Teperson advocated excision of the lesion while others recommended non-interference to deep X-ray therapy. We feel that to promote healing, minimal doses of deep X-rays as was done in our case is desirable. Given time and rest to the part the lesion appears to be self limited.

Summary

1. Literature regarding eosinophilic granuloma of bone is briefly reviewed.
2. Clinico-pathological findings in a case of eosinophilic granuloma of femur are recorded and attention of the surgeons is drawn to the regression of the lesion by deep X-ray therapy.
3. The utmost necessity of biopsy examination in the diagnosis of the condition is stressed.

Acknowledgment

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PARAPLEGIA DUE TO AORTIC ANEURYSM

BY

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Paraplegia caused by an aneurysm of the aorta is not a common condition. Only a few cases are reported in the literature. Laennec[1] in 1925 reported the first case. Shimkin[3] has summarised the literature on the subject. In India Nimbkar and Ginde[2] have reported two cases.

An aneurysm of the aorta may cause paraplegia either by direct pressure or by interference with the blood supply of the cord or both. Paraplegia due to interference with the blood supply of the cord is perhaps commoner. In an expanding aneurysmal mass, the process of thrombosis in the walls may involve the mouth of a series of intercostal arteries and occlude them. This diminishes the blood supply of the concerned segments of the cord. Occasionally when an aneurysm starts by tearing the middle coat of the aorta and causes a dissecting aneurysm there may be an acute diminution or loss of blood supply to the cord with onset of rapid paraplegia.

Direct compression of the cord by aortic aneurysm is not very common. The expanding pulsating mass causes severe pressure errosion of the vertebra. The intervertebral cartilages are not so much affected as the vertebrae. Rarely a vertebra may be completely eroded and the aneurysm come into direct contact with the anterior or anterolateral aspect of the cord and compress it.

A careful neurological examination and a study of the cerebrospinal fluid help to differentiate between cord compression and paralysis due to insufficient vascular supply.

Case Report

Mr. A., aged 48 years was admitted with a complaint of weakness of his legs and inability to walk. Three years ago while working on night shift he had a sudden attack of pain in the back in the upper dorsal region. This was severe and localised and lasted only for a few minutes. Similar attacks of pain used to come on occasionally for the past two months. About three months later he noticed weakness of his left lower limb, which gradually increased in severity causing him to limp. The right side was affected in a similar manner later. Next year the patient was unable to walk. A progressively increasing kyphosis in the upper dorsal area was also noted by him. This was not accompanied by pain either localised or radiating.

The patient had not experienced at any time any shooting pains along his arms or legs, or any associated weakness of the arms. Neither did he give any history of trauma to the spine or a history of venereal disease.

On examination the spine showed a kyphotic deformity in the upper dorsal area which was neither tender nor painful on movement.

The upper extremities were not affected in any way. The lower extremities showed complete loss of power and flaccid tone, without any involuntary movements or any nutritional changes in the muscles or the skin. Sense of touch was lost upto the 7th dorsal on the right side and 5th dorsal on the left. Pain was impaired to similar levels. Both vibration and joint sense were not appreciated and no exact level of hyperaesthesia was noted.

The deep reflexes were exaggerated in both legs. Ankle clonus was present but was not sustained. The plantars were not elicited. The cremasteric and abdominal reflexes were lost on both sides. The bladder was found to be distended and there was overflow incontinence.

Examination of the chest showed dullness in the mid zone on both sides. The patient did not have any pressure symptoms like dyspnoea or dysphagia. The blood pressure recordings from all the limbs were normal.

The blood W.R. and Kahn were found to be negative. X-Ray of the chest showed a mass in the mediastinal region (Figs. 1 and 2) and the lateral view showed clearly, erosion of the bodies of the vertebrae. (Fig. 3).

A diagnosis of an aneurysm of the aorta causing paraplegia was made. Lumbar puncture was done. This showed complete block in the subarachnoid space with 400 mgs. of proteins in the crebrospinal fluid.

The patient had already had elsewhere a full course of anti-syphilitic treatment in spite of blood W.R. being negative. Hence it was felt that this was probably not a syphilitic, aortic aneurysm but a dissecting aneurysm of the aorta specially from the history of the periodic attacks of severe recurrent pain in the back. As no other treatment was thought feasible and as an exploratory measure, it was decided to try the effect of spinal decompression. Laminectomy of 3rd, 4th, 5th and 6th dorsal vertebra was done. The laminae were found to be very thin. The mass was found lying just anterior to the spinal cord which appeared tape like, being thinned out antero posteriorly. Postoperatively the patient did not have any improvement except slightly better appreciation of sensation. The patient was discharged. Nine months later the patient complained of another attack of sudden severe pain in the back and the chest and expired. No post-mortem was available.

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Fig. 1
P.A. view of chest showing a big mediastinal mass.

Fig. 2
A.P. view showing the aneurysm extending almost down to the 11th dorsal vertebra.

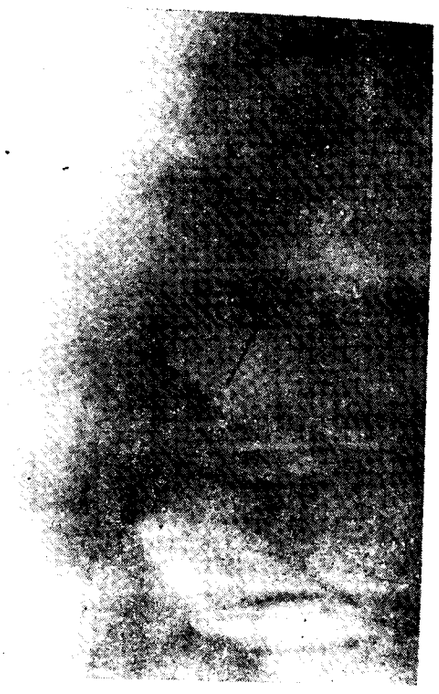

Fig. 3
Lateral view of spine showing erosion and destruction of the vertebra.

Discussion

The paraplegia in this patient was caused both by direct pressure on the cord as well as by interference with the blood supply of a large number of spinal segments. The presence of flaccidity in the paralysed legs suggested that an extensive portion of the spinal cord was involved. Compression of the cord was proved by lumbar puncture as well as by surgery. The interference with the vascular supply was shown by the neurological signs.

Summary

Paraplegia caused by an aneurysm of the aorta is not a common condition. A case is reported in which paraplegia was caused by a dissecting aneurysm of the aorta pressing on the cord and also interfering with the vascular supply.

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AINHUM

(DACTYLOLYSIS SPONTANEA)

BY

V. S. CHINCHOLKAR, NAGPUR

Ainhum (Dactylolysis Spontanea), with a dozen synonyms, is a chronic slowly progressive tropical or semi-tropical disease, affecting the toes, particularly the fifth toes and more rarely the fourth and other toes, of dark-skinned races both in the old and new world. It is characterised by the formation of a furrow at the digito-plantar fold, which deepens and extends until it finally encircles the toe. If allowed to proceed without surgical interference it eventually results in spontaneous amputation. The term is derived from two sources : The African Nago dialect of the Yoruba Language meaning 'to saw or cut' and the Brazilian Negro Patois meaning 'fissure.'

History : Messum is credited with the first report of this disease (1821), while da Silva lima described the condition in 1852 as occurring in Brazil and christened it 'AINHUM' in 1867. Clarke (1860) in his description of the condition in Gold Coast considered it to be a manifestation of Yaws. In 1873, Crombie described the disease as occurring in India. Duhring in 1884, studied the disease microscopically considering the essential pathological feature to be an inflammatory oedema of the hypodermis. In 1886, Engles describing the pathology of the condition concluded that irritation caused an internal proliferation of the epithelium, which extending into the cutis, damaged the vasomotor nerves, leading to a spasm of the vessels, endarteritis obliterans, fibrosis of the cutis, and rarefying osteitis, whereby the digit is separated from the foot.

Incidence : The disease occurs in South America especially in Brazil and Argentina, in North America especially in Southern States, but also, though rarely in the Northern States, and in Canada. It also occurs in the West Indies. In Africa it is especially well known on the west coast and particularly in the Gold Coast. In Asia it is known in India, (?) China and Ceylon. It has been seen occasionally in Southern Europe especially Italy.

Aetiology : The aetiology of the disease is not known ; but many theories have been expounded as to its cause. da Silva lima observed the disease in all the members of a negro family and believed that heredity might be a factor. Unna and others noticed a close association to Scleroderma. Wucherer and others considered the condition to be a trophoneurosis. Eyles believed that the fibrotic constriction was secondary to mechanical or infectious injury. Pusey and Castellini inferred that the disease was parasitic in origin. Shaffer reported a case in which diabetes and arteriosclerosis may have been the cause. Others have blamed syphilis, yaws, leprosy, constricting rings, bands or strings. Zambacho Pasha, Engles and Moreira are of the view that the reason why the native races are attacked is because they walk barefoot and that irritation or injury to the skin of the little toe is more likely to occur. It is more common in males than in females, in adults than in children and is more common between 30 to 35 years. It runs its course in from one to ten years or even more.

Symptomatology : The disease commences as a narrow groove in the skin, almost invariably on the inner and plantar side of the root of the little toe. It may occur in one foot only or in both feet simultaneously, or it may affect one foot after the other. The groove, once started, deepens and extends gradually round the whole circumference of the toe. As it deepens, perhaps, though not necessarily with ulceration, the distal portion of the toe is apt to swell to a considerable size, as if constricted by a ligature. There is little or no pain, although there may be inconvenience from the liability to injury to which the dangling and everted toe is exposed. Often an ulcer forms on the inner side of the toes and causes much pain. In the course of years the groove slowly deepens and finally the toe drops off or is amputated. The groove may or may not correspond to a joint. In rare instances, after the two distal phalanges have dropped off, or been amputated, the disease occurs in the stump and the proximal phalanx in its turn is thrown off. Of the other toes, the fourth is the one which is most frequently affected, very rarely is the third, second

or great toe affected. Occasionally the terminal phalanx of the fifth digit of the hand has been affected.

Histopathology : The constant irritation causes the epithelium to proliferate internally and depress the skin and cause the fibrous tissue of the cutis to proliferate. There is also an endarteritis, by which the blood supply is gradually cut off the distal portion of the toe, which therefore becomes oedematous and fatty, while the bone undergoes rarefying osteitis, so that the digit is gradually separated from the foot, a process which takes place through the bone of a phalanx. The histological examination at the line of the furrow shows proliferation of the epidermis, which projects downwards into the cutis, in which the connective tissue is increased in quantity. The vessels show endarteritis and periarteritis, the sweat glands show proliferation and fatty degeneration of the cells. The bone is in a condition of rarefying osteitis. Distal to the furrow the joints are effaced, the tissues show fatty degeneration and oedematous infiltration. No organisms can be found.

Case Report

Two cases were observed and are being presented.

Case I

Adult 25, H.M. presented with complaint of pain in both fourth toes since 1941. He was a police constable prior to his disability but had to leave the force because of his inability to put on boots. Family history—Nil, particular. No past history of syphilis. Habit of walking bare-foot.

General examination showed no clinical evidence of Leprosy or Yaws.

Local examination revealed annular constriction around the bases of the fourth toes, going all round the circumference. Parts distal to the constriction were swollen but there were no colour changes. Constriction was just appearing on the dorsum of the right fifth toe. Left fifth toe, no constriction seen. (Fig. 1).

No evidence of interdigital fungal infestation. X-ray of the foot showed no evidence of bony changes.

Blood for K.T. negative.

Treatment : Both the fourth toes were amputated because the patient was getting unbearable pain.

Gross features show rings of constriction at the posterior ends of both toes. (Fig. 2).

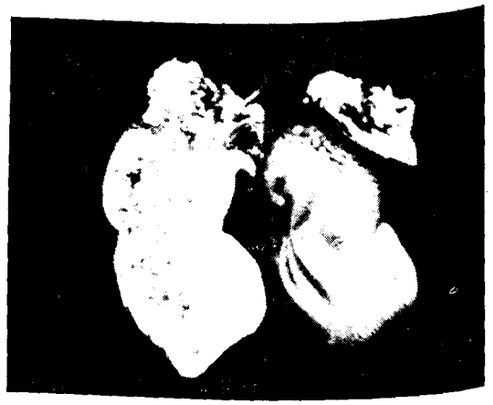

Fig. 2
Shows rings of constriction at the top of both amputated toes.

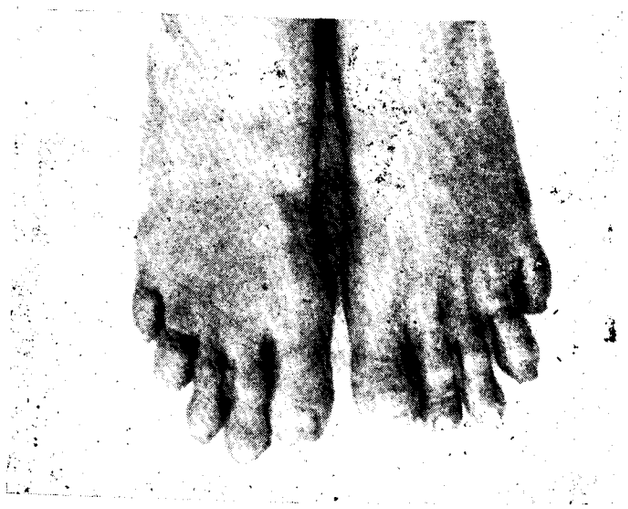

Fig. 1

Shows rings of constriction around the bases of both 4th toes and on the dorsum of right 5th toe.

Histopathological examination : shows hyperkeratosis of the skin. At one place there is sudden constriction in this layer with changes of degeneration. The epidermis underneath the constriction is atrophic. Dermis is normal. Blood vessels show thickening of intima. Bone underneath is normal. No changes of inflammation seen. The picture is that of localised atrophy of layers of the skin. (Figs. 3 and 4).

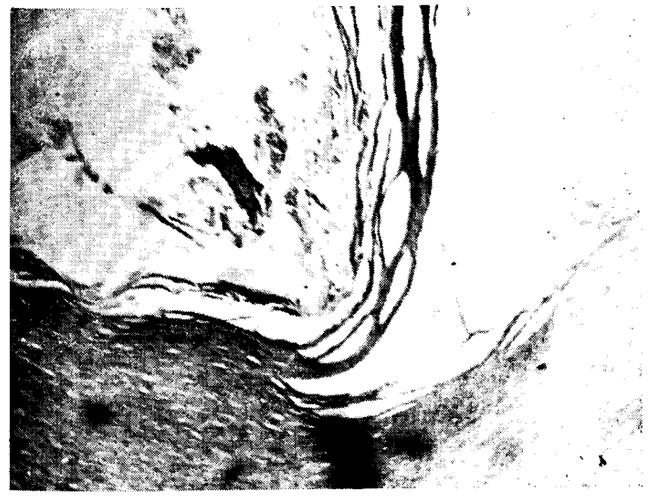

Fig. 3

Shows hyperkeratosis of the skin with sudden constriction in this layer at one place.

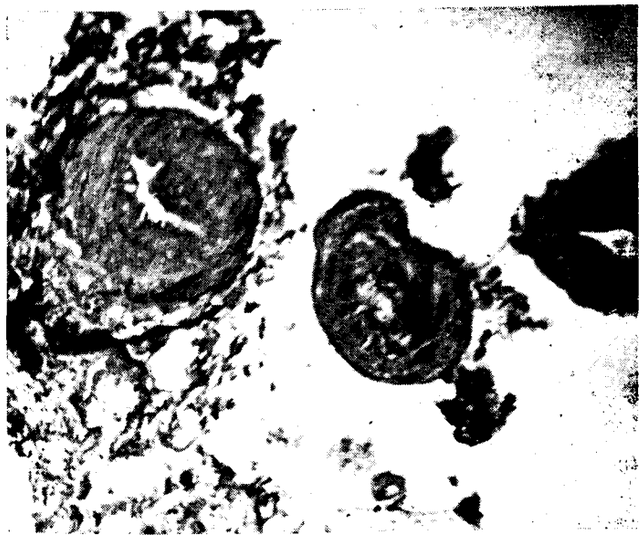

Fig. 4

Shows thickening of the intima.

Case II

Adult 40 H.M., a farmer complained of rings of constriction around the left fourth and fifth toes (fig. 5). Right fifth toe was amputated for the same complaint three years ago. Interdigital fungal infestation was present. Habit of walking bare-foot. No clinical evidence of Yaws or Leprosy. Blood for K.T. Negative. X-ray showed rarefying osteitis in the bone of left fourth and fifth toes. Patient refused operation and hence histopathological study of this case could not be made.

Fig. 5
Shows rings of constriction around the bases of left 4th and 5th toes.

Observations

In cumulative index from 1938 to 1953 there are 45 articles on Ainhum but all of them from countries outside India. In 1873, Crombie reported cases occurring in India. Unfortunately no case reports are available to indicate as to in which parts of our country this disease is prevalent. Two cases are being reported as occurring in these parts and observations by other workers about the prevalence of the disease in their areas would be welcome, so that they may bring out some common factor, which may be of aetiologic significance.

Summary

1. Two cases of Ainhum are presented.
2. The disease is described in detail.

Acknowledgment

I am grateful to Dr. J. B. Shrivastava, Prof. of Pathology for the histopathological report and microphotographs, to Dr. R. Nigam, Prof. of Surgery, for his guidance and to Dr. P. M. Bhandarker, Dean, Medical College and Hospital, Nagpur for permission to publish this article.

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PROLAPSE OF ILEUM THROUGH A PATENT VITELLO-INTESTINAL DUCT

(A Review of the literature with a report of 2 personal cases)

BY

WARYAM SINGH AND I. C. PATHAK AMRITSAR

Persistence and patency of the vitello-intestinal duct is a rare congenital anomaly (Fig. 1) At St. Lake's Hospital, Cleveland, among 31,975 births over a period of 21 years, 2 babies were born with complete patency of the vitello-intestinal duct, approximately 1 in 16,000 or 0.0063% (Brown & Glover 1952). The first report of a patent vitello-intestinal duct was by Poussin in 1817 (Arnheim 1950). Prolapse

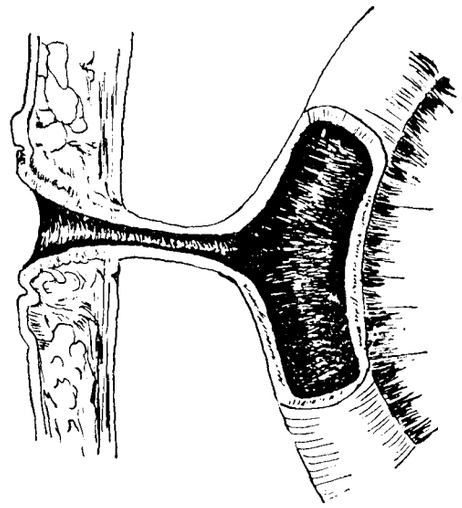

Fig. 1
Diagram of Patent Vitello-Intestinal Duct, showing the lumen of the duct directly continuous with that of the ileum
(*Arnheim, Ann. Surg.* 91 : 77, 1950)

of the small gut through a patent vitello-intestinal duct on to the abdominal wall is a still rarer condition with a high fatality rate. Such a prolapse or intussusception has been described as a dramatic and terrifying occurrence (Moore 1956). Till 1956, there were reports of 45 such cases in the literature with only 12 recoveries. In this report we present two cases of prolapse through a patent vitello-intestinal duct which were operated upon successfully.

In the 3rd to the 4th week of intra-uterine life, the primitive mid-gut is connected to the yolk sac of the embryo by the medium of the vitello-intestinal duct. A gradual and progressive obliteration of the lumen of this duct begins during the 5th week and is usually completed by the 7th or 8th week of intra-uterine life. The duct, however, remains open in a small number of cases to produce different varieties of congenital anomalies depending upon the degree of patency. Brooks (1954) describes the following patterns :

(1) In about 80 per cent, the anomaly is of the type described by Meckel in 1816. The classical Meckel's diverticulum arises from the anti-mesenteric border of the ileum about 2 feet from the ileocaecal valve and is about 1½″ to 2″ long. The end is closed and rounded and is free of umbilical attachment. The histological structure of its wall is similar to that of the small gut.

(2) In about 10 per cent, the diverticulum resembles the type described above but has a fibrous cord attached to its apex. The fibrous cord may lie free in the peritoneal cavity or gain attachment to the umbilicus or any other abdominal viscus.

Meckel's diverticulum has been reported to occur in 1–3 per cent of the population.

(3) In about 6 per cent, the whole of the vitello-intestinal duct remains patent so that there is a canal running from the small gut to the umbilicus resulting in a fistula. The patent duct may be quite short so that the small gut may be attached directly to the posterior aspect of the umbilicus, as was seen in our 2nd case.

(4) The rest of the anomalies are rare and are comprised of—

(*a*) formation of an umbilical sinus if only the distal end of the vitello-intestinal duct remains patent.

(*b*) formation of a cyst, if the two ends of the vitello-intestinal duct close and only the central part remains patent.

(*c*) formation of a polyp at the umbilicus as a result of the persistence of a small area of

mucous membrane of the vitello-intestinal duct at the umbilicus.

A persistent and patent vitello-intestinal duct presents itself clinically as a red nodule of mucous membrane at the umbilicus with a fistulous opening in the centre exuding intestinal contents. The most satisfactory diagnostic procedure to visualize the patency of a persistent vitello-intestinal duct is by X-Ray examination after injecting a radio-opaque dye into the fistulous opening at the umbilicus. IAN AIRD (1957) is of the opinion that in some cases, the fistula may be a safety valve to a distal stenosis in the small gut.

In a case of prolapse there is usually a single or T-shaped protruding loop of small intestine through the umbilical region (Figs. 2 and 3). The surface of the everted mucous membrane is dark red in colour. Some degree of shock is usually present. The prolapse can be reduced if it is small and early. Surgical intervention then comprises excision of the duct and part of the small gut with anastamosis of the resulting two ends. If the prolapse is not reducible excision of the segment of small gut, including the duct and prolapse, is carried out with end to end anastamosis, as was performed in our first case. If the duct is small or

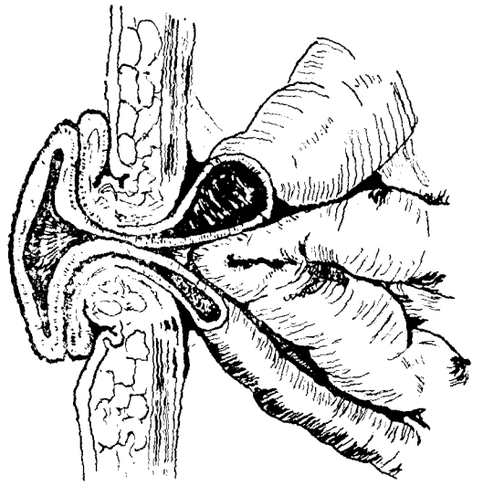

Fig. 2

Diagram of prolapse of ileum through a patent vitello-intestinal duct showing a sausage-shaped mass lying on the surface of the abdomen covered with mucosa of ileum. It has an upper and a lower opening corresponding to the lumen of the proximal and distal loops.

(*Arnheim, Ann. Surg.* 91 : 77, 1950)

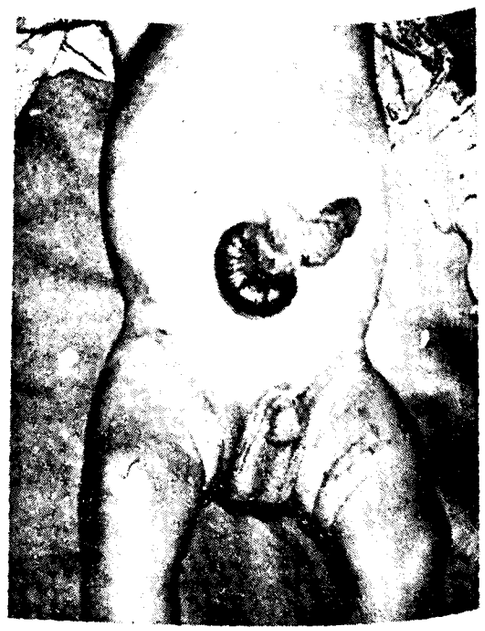

Fig. 3

Case No. 2. Pre-operative photograph showing T-shaped prolapse of ileum through a patent umbilicus. The mucous surface shows discolouration, the colour returning to normal after reduction of prolapse.

non-existent, as in our 2nd case, simple closure of the defect in the wall of the small gut may suffice, after reduction of the prolapse.

Morgan in 1942 reviewed the literature and collected 105 cases of patent vitello-intestinal duct with prolapse of small gut in 26 cases. Kittle et al (1947) collected 128 cases of patent vitello-intestinal duct recorded in the literature prior to 1947. They added 2 cases of their own and one observed by Morgan of Cleveland (Brown and Glover 1952) Brown and Gain (1950) reported 1 case of prolapse being the 28th recorded instance of this type. Of these 28 cases, 14 were not operated upon. Two reduced spontaneously with survival and of the 14 operated upon only 4 survived. Moore (1956), however, reports that the literature concerning intussusception of ileum through a patent vitello-intestinal duct was reviewed by Moore and Shumacker in 1952 and up to that time only 33 cases had been reported and only 3 of the patients survived. He further reports that since 1952 reports of 7 additional cases have appeared in the literature. Five

of these patients recovered. Moore (1956) reports 5 additional cases of prolapse. Operative reduction of the intussusception followed by the resection of the vitello-intestinal duct was carried out in 4 cases and all these, with the exception of the fifth patient with congenital heart disease who was not operated upon, recovered.

Better operative results during the last 5 years appear to be due to prompt surgical relief, recent advances in anaesthesia, availability of blood for transfusion, the use of antibiotics and a better understanding of fluid and electrolyte balance.

Two cases of prolapse of the small gut through a patent vitello-intestinal duct were treated by us by early operative intervention. Both the patients survived. In the first case a 2 stage resection and end to end anastomosis was performed, while in the 2nd case, manipulative reduction having failed, the T-shaped prolapse was easily reduced after division of the constricting band at the umbilicus. It is of interest to note that the first case, a 20 days old male baby, presented as an emergency with a prolapsed gut of 2 days' duration. No red mass or discharge at the umbilicus was noticed by the parents before this. The 2nd case was admitted as a case of faecal fistula, at the umbilicus and the prolapse occurred in the ward while awaiting operation.

Case 1

J.S., 20 days old, male baby was admitted in the East Surgical Ward of the V.J. Hospital, Amritsar on 9th August 1955 with the history that 2 days prior to admission the umbilicus had suddenly burst with protrusion of gut through it with faecal discharge. The baby vomited repeatedly and did not pass faeces per anus. The parents had not noticed any red mass or discharge at the umbilicus before the sudden rupture. Examination revealed a T-shaped protrusion of gut through the umbilicus, the mucous membrane of which was dark red in colour. There was mucoid discharge from this surface and small bits of faecal matter were sticking to the abdominal wall around it. Manipulative reduction was not considered feasible because of the time lapse and marked colour changes of the prolapsed gut. Subcutaneous drips of 2½% glucose and ¼ strength saline was started and a rubber catheter was introduced intranasaly for gastric suction. Under open ether anaesthesia a mid line incision surrounding the umbilicus was made. The ileum proximal to prolapse was moderately distended while the distal ileum was collapsed. The returning layer had burst through the ensheathing layer at one place. The whole mass was excised between clamps. Baby's condition at this stage was reported to be very poor, so both the clamped ends of the ileum were exteriorized through the lower part of the wound, the rest of which was closed in layers. Two days later the baby's condition was quite satisfactory and under open ether anaesthesia, end to end anastomosis of the exteriorized ends was performed. The subcutaneous drip and intra-nasal catheter were maintained for another 2 days while penicillin, streptomycin, Vit. B complex and Vit. C were administered parenterally. He made an uneventful recovery and was discharged on the 9th day after the 2nd operation.

Case 2

G.S., 39 days old baby, was admitted in East Surgical Ward on 11th March 1957 with a red mass about 1 cm. in diameter over the umbilical region of about 34 days' duration. The parents first noticed this mass on about the 5th day after birth when the umbilical cord fell away. The baby was first seen in the surgical outpatient when he was 22 days old and diagnosed as a case of patent vitello-intestinal duct with a faecal fistula. The parents were advised to bring the baby for admission on 11th March 1957.

On admission the general examination revealed no abnormality. On local examination a red mass of granulation about 1 cm. in diameter was seen at the umbilical region with a small opening at its centre. Faecal discharge came out of this opening whenever the patient strained. There was no history of vomiting and the bowel function was normal. On 16th March 1957, barium meal study of the gastro-intestinal tract was carried out to rule out the presence of stricture beyond the fistulous opening in the small gut. No abnormality was detected. On 17th March 1957 at about 5 p.m., the mother noticed a sudden increase in the size of the mass at the umbilical region. On examination prolapse of the small gut mucosa was noticed forming a T-shaped loop about 4" long lying trans-

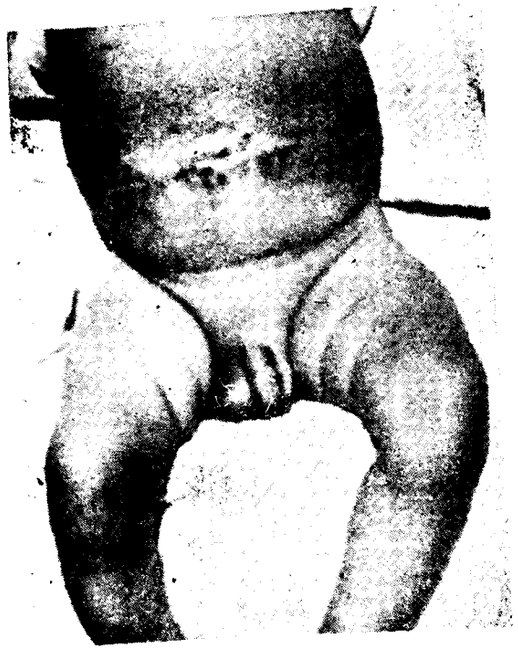

Fig. 4

Case No. 2. Post-operative photograph showing primary union of operation wound.

versely with openings at both of its ends (fig. 3). The general condition of the baby was good. Manual reduction of the prolapsed mucosa was tried but was not successful. At 7-15 p.m. under semi-open ether anaesthesia a transverse elliptical incision was made surrounding the umbilicus. On opening the peritoneal cavity a loop of small gut was found to have prolapsed in a T-shaped manner through the patent umbilical opening. Manual reduction of the prolapse was possible only after dividing the constricting ring at the umbilicus. The opening in the small gut was freed from the umbilicus, to which it was almost directly adherent. This opening was closed in a transverse line in 2 layers. Wound was closed in layers without drainage. 2½ per cent glucose in normal saline was started intravenously at the outset of the operation and, along with gastric suction through an intra-nasal catheter, was maintained till the 3rd post-operative day. Parenteral administration of penicillin and streptomycin was started on the day of operation but was omitted next day and replaced by Terramycin pediatric drops. Wound healed by primary intention (fig. 4). Patient was discharged on 30th March 1957.

Summary

Patent vitello-intestinal duct with prolapse of the small gut through the umbilical region is a rare congenital anomaly. Before 1952, 33 cases had been reported in the literature with only 3 survivals. During the last 5 years, reports of 12 more cases have appeared in the literature with 9 recoveries. Authors present a report of 2 cases which were treated by early operation. Both the patients survived.

Acknowledgment

Our thanks are due to Dr. S. S. Anand, F.R.C.S. (Eng.) F.A.C.S., F.C.C.P., Professor of Surgery, Medical College, and Medical Superintendent, V. J. Hospital, Amritsar for permission to publish these cases.

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TUBERCULOSIS OF THE PANCREAS

(A Case Report)

BY

S. S. ANAND AND WARYAM SINGH, AMRITSAR

Tuberculosis of the Pancreas is an extremely rare condition of which only a few cases have been reported in the literature. Involvement of Pancreas is very uncommon even in Military Tuberculosis (Anderson). We have not been able to find any reference to this disease in the surgical literature available to us. It is of interest, therefore, to report a case of Tubercular Pancreatitis proved on Biopsy.

Case Report

J.D., 45 years old female was transferred to the East Surgical Unit of V.J. Hospital, Amritsar, on 14th September 1956, from the medical side, where she had been admitted earlier for investigations of a mass in the right hypochondrium and epigastrium. She had a history of recurrent attacks of colicky pain in the right hypochondrium, radiating to the back of 2 years' duration. This pain had become almost continuous since 2 months before admission. The attacks were accompanied by a few vomits, but no fever or jaundice. During general examination two firm mobile glands were palpated in the lower part of the left side of the neck. Examination of the abdomen revealed tenderness over the right hypochondrium and epigastrium. An indefinite mass which moved slightly with respiration, was palpable in the right hypochondrium.

Investigations :

Haemoglobin	:	10.5 Gm.%
Total Leucocyte count	:	9700/cc.
Diff. Leucocyte count	:	68, 28, 4, 0.
E.S.R.	:	98 m.m. first hour (Westergreen).

Stools, screening of chest, radiogram of chest and plain radiogram of abdomen did not reveal any abnormality.

Liver function tests—normal values.

Oral Cholecystography—Gall bladder not visualised. Barium meal study of stomach and duodenum did not show any abnormality.

Laparotomy was performed on 20th September 1956. The gall bladder was found to be moderately distended, had thick opaque walls and enlarged cystic gland. No stones were palpable. There were fine patches of fat necrosis covering the duodenum, pylorus and the free edge of the lesser omentum. While palpating the lower part of the common bile duct, it was found that the pancreas was enlarged and firm. The enlargement was uniform in the whole of its extent, as confirmed by opening the lesser sac. A biopsy of the pancreas was taken. Cholecystectomy performed. Common bile duct was opened and found to be quite patent, allowing a free passage of 9/12 urethral bougie into the duodenum. The common bile duct was drained by a T-tube, which was brought out of a separate stab wound, along with a drainage tube from Morrison's pouch. Wound was then closed in layers.

Injections of Penicillin and Streptomycin started pre-operatively, were continued. Post-operative period was uneventful till the 7th day when stitches were removed. The wound had healed by primary intention. The T-tube was draining well.

The histopathological report of the excised gall bladder, received at this stage was Chronic Cholecystitis. The biopsy from Pancreas showed the typical caseation necrosis and giant cell formation of Tuberculosis. (Figs. 1 and 2).

It was now proposed to remove one of the cervical glands for histopathology, but on the 9th post-operative day the patient complained of mild pain in the epigastrium, where a diffuse firm mass was palpable. By the evening she was in a collapsed state, with a rapid thin pulse, low blood pressure and slight cyanosis. She was put on I. V. 5% glucose drip, achromycin, cortisone 25 mgm. T.D.S. and oxygen inhalation. It was

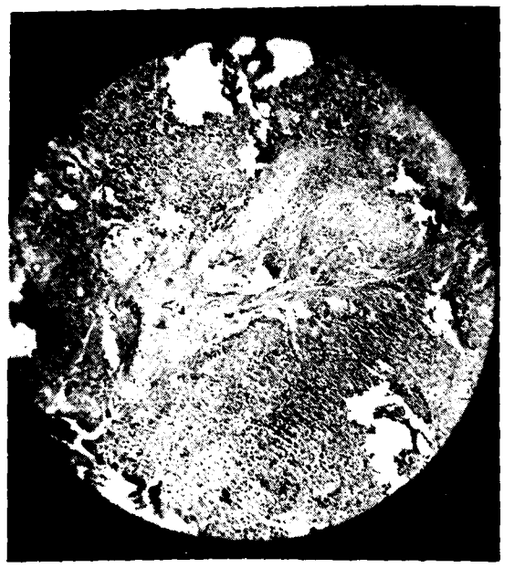

Fig. 1

Pancreatic biopsy, low power, showing areas of Caseation necrosis and giant cell formation.

Fig. 2
Pancreatic biopsy, high power, showing typical giant cells of Tuberculosis.

thought that she had suffered an attack of acute Pancreatitis. A specimen of blood was taken for serum amylase estimation but unfortunately became haemolysed. Fasting blood sugar was 80 mgm. %. She rallied round by the 2nd day of this acute episode, but was later removed from the hospital against medical advice. An attempt to follow-up the patient has not been successful.

This rare case of Tuberculosis of the Pancreas is also of interest in that it was not associated with any tubercular lesions in the lungs or abdomen, though possibly glandular tuberculosis was present in the neck. Even on laparotomy the tubercular nature of the lesions was not suspected. The presence of fat necrosis and uniform firm enlargement of the pancreas was at that time thought to be the result of relapsing Pancreatitis and the common bile duct was opened with a view to assess the state of the sphincter of Oddi. In the absence of pancreatic biopsy, the true diagnosis would certainly have been missed. A needle biopsy probably have been a safer procedure (Kirtland, 1951) but could not be employed, since a Vim-Silverman needle was not available in the operation theatre.

Summary

A case of Tubercular Pancreatitis is presented. It is suggested that pancreatic biopsy be more frequently employed in cases of demonstrable pancreatic enlargements.

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NEUROBLASTOMA SYMPATHETICUM

BY

JAYANT M. JALUNDHWALA AND R. C. SHAH, AHMEDABAD

The commonest lesion of the spine that an orthopedic surgeon meets with, in children, in this country, is tuberculosis of the spine. But the case presented herein, shows that the unusual condition of Neuroblastoma Sympatheticum must be kept in mind, before one makes a final diagnosis.

Neuroblastoma Sympatheticum is the second commonest tumour among the retroperitoneal tumours of children (Gross), the first being Wilm's tumour. Other uncommon tumours are retroperitoneal teratomas and a few miscellaneous ones. This tumour arises mainly in infancy, 80% cases being noted in the first five years of life. The distribution of the lesion in the two sexes is almost equal. The commonest site is the adrenal medulla. Abdominal sympathetic chain is the second commonest situation in the abdomen. Other situations where it has been known to occur are Thoracic sympathetic chain, Cervical region (Stewart 1919), Scapular region (Symmers), Presacral sympathetic fibres (Harbiz 1915) and region of the intervertebral foramen (Cushing and Walbach 1927). The tumour makes its presence felt essentially by the mass, or by the presence of secondaries. A large number of cases, (about 60%) reported in the literature, already had secondary deposits at the time of the first clinical examination (Gross). The clinical picture with which a patient would present, would therefore naturally depend upon the situation of the original tumour and on the presence of metastases. The case that is being presented here, had paraplegia and a firm mass in the right loin when first seen. It has been noted that quite a few cases, when first seen, present themselves for the secondaries, the primary not being noticed. The tumour of the right adrenal gland is known to give rise to large secondaries, mainly in the liver. This syndrome was known as the Pepper syndrome. The left adrenal gland tumour usually gives rise to secondaries in the skeletal system *viz.* skull, vertebra, ribs, pelvis, etc. This syndrome is known as the Hutchinson syndrome. A third type was also being described namely Esser–Herwig type, wherein there was an association of bony metastases with leucoerythroblastic anæmia. However, Karsner (1950) has observed, of late, that these different syndromes of the same disease process cannot be separated out as different entities : although the different syndromes may have a different prognostic value : the pepper syndrome for example has a better and a longer survival rate than the Hutchinson syndrome.

These tumours are very malignant tumours, though a few of them are of comparatively low malignancy. A few of these tumours have been reported to have lost their malignant character by the occurrence of spontaneous haemorrhage. Three such cases have been reported by Gross. Usually the children affected die due to extensive metastases, and treatment however energetic, has been quite often very disappointing. An occasional complication, causing the death of the patient has been intraperitoneal haemorrhage as reported by Gorgono (1956).

The tumours essentially arise from the cells that go to form the adrenal medulla and the sympathetic system. The parent cell, the sympathagonia, is a small spherical cell, 7–8 microns in diameter, with densely staining nucleus and very little cytoplasm. This cell later develops into a sympathoblast, which is larger, 3–12 microns in diameter, and has a pale vesicular nucleus with relatively more cytoplasm and the presence of fibrils. This can migrate into various regions, to form the anlage of the sympathetic system. The next stage, in the development, is the formation of the ganglion cell. This is a large and irregularly shaped cell with many argentophil cytoplasmic processes.

The malignant tumour may develop at various stages of development. The degree of malignancy however depends upon the cell-type : tumours with more immature and dedifferentiated cell-type being more malignant. The same tumour may have different cell-types due to different grades of dedifferentiation being present. Even so, certain well-formed tumourt ypes are recognised depending upon the predominant cell-type present. The sympathogonioma is the tumour having the most premature cell-type, and may appear very much like a small-round-cell-sarcoma showing densely packed r and

cells with very little cytoplasm and scanty connective tissue. The sympathicoblastoma, however presents a fair number of pseudorosettes of cells with demonstrable fibrils being present.

Although in the past the therapy of these tumours has been very unsatisfactory and depressing, the newer forms of therapy, introduced by Lehman, Farber and, of late by Wittenborg has changed the prognosis of these tumours to a certain extent. The last author has recorded certain successful forms of post-operative radiotherapy for use in such cases. The three-year cure-rate has improved from 9%, before 1940 to 29% in 1947 (Gross) and onwards. In certain series, a higher cure-rate of 45% also has been obtained.

The essential treatment is therefore outlined by a combined attack, Surgical and radiotherapeutic. As complete an excision as possible, is made, to be followed by radio-therapy. Recently, however, a report has appeared on the treatment of these cases by massive doses of vitamin B_{12} 1000 mcgms on alternate days. This method of treatment has shown a definite regression in 10 cases. Since the series reported is too small it is too early to procrastinate on such a therapy.

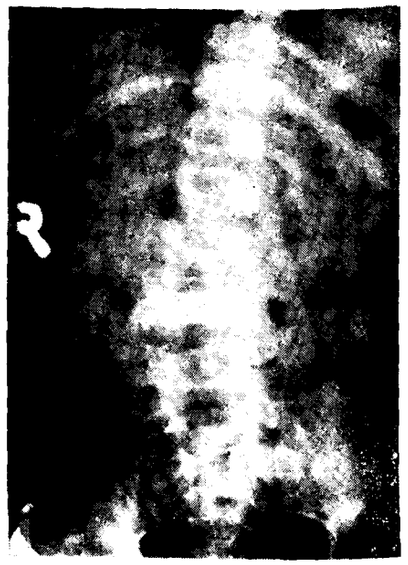

Fig. 1

Skiagram of the lumbar spine, postero-anterior view showing sclerosis of the third lumbar vertebra with flattening and fine granular calcification in the soft tissue shadow, to the right of the third lumbar vertebra.

Case History

A.N., Hindu male child, aged 6 years, was admitted to the hospital with the complaints of deformity of lumbar spine, swelling in the right loin, and paraplegia of 7 months duration. There was a history of fall, while walking, on two occasions, a month prior to the onset of the above complaints. The paraplegia was noted after the second fall. The child was seen and treated at two other hospitals, in the month of August 1955 and December 1955, respecitvely. The diagnosis, considered there, was tuberculosis of the spine and hence was advised anti-tuberculous drugs and immobilisation in posterior plaster shell.

Clinical examination revealed left-sided scoliosis, with loss of normal lumbar lordosis and a very firm, non-tender, non-fluctuating mass, 5″ × 3″ in size, in the right loin, fixed to the posterior abdominal wall. The mass was also palpable in the right lumbar region, but was not ballotable. Skin over the swelling was normal and there were no prominent veins. There was no evidence of free fluid in the abdomen. Liver and spleen were not palpable. No other masses were felt in the abdomen. Urinary bladder was distended upto umbilicus, but was not tender. Rectal examination was normal.

On general examination the boy looked thin and anæmic. There were no enlarged glands in the cervical, axillary or inguinal regions.

C.N.S.

On examination of the nervous system, it was found that motor power was diminished in the left lower extremity and was completely lost in the right lower extremity. There was hypotonia in both the lower limbs. There was generalised wasting in both lower limbs, more on the right side than on the left and more of the leg muscles, than of the thigh muscles. Sensations were all present. Reflexes: Superficial abdominal and cremasteric reflexes were present on both the sides. Planter reflexes could not be elicited on either side. Knee jerk on the right side and ankle jerks on both the sides were lost. Knee jerk on the left side was sluggish. There was retention of urine. Defaecation was normal. Total neurological deficit suggested partial pressure on the cauda equina, more on the right than on the left side.

Heart and lungs were normal.

Investigations

On admission haemogram revealed R.B.C. 4.03 million/c.m.m., Hb. 7.4 gms. Colour index 0.66, W.B.C. total 9800/c.m.m. Polymorphs 24% lymphocytes 73%, eosinophils 1%, monocytes 2%. Blood group 'O.'

Skiagram of the lumbar spine in Posteroanterior and lateral positions showed marked sclerosis and slight flattening of the body of the 3rd lumbar vertebra. The laminae were destroyed on the right side but the intervertebral discs were normal. There was a visible soft tissue shadow to the right of the lumbar spine, showing finely granular calcification. (Figs. 1 & 2).

An incisional biopsy of the mass was done on 27th February 1956. This was reported by the Pathologist as Neuroblastoma Sympatheticum.

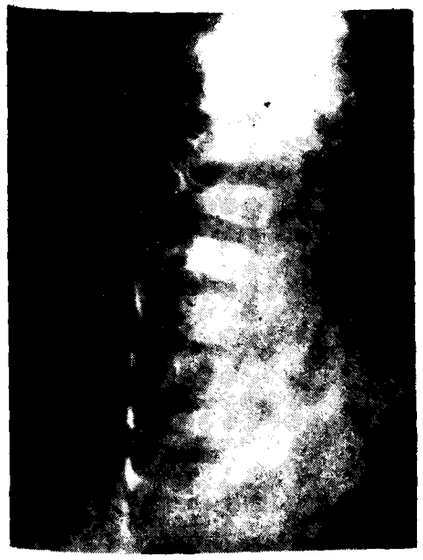

Fig. 2

Skiagram of the lumbar spine, lateral view showing sclerosis of the third lumbar vertebra with destruction of its lamina. The intervertebral discs are normal.

Pathological Report

The sections show infiltrating tumour mass, arranged in solid cords, separated by poor septa of connective tissue. The tumour has a diffusely cellular undifferentiated pattern. The tumour cells are undergoing necrosis and there is leucocytic infiltration in occasional necrotic foci. The tumour cells are round, polygonal, or pear shaped. The cells consist almost entirely, of the nucleus, which stains hyperchromatic. Mitotic activity is seen. Nuclear size and ratio is variable, within some limit. There are no giant cells. The cellular processes are not evident, but at places there is formation of typical rossettes. Blood vessels are supported by tumour cells, and have poorly developed walls. There are many nerve fibres in and around the tumour. The histology is that of a Neuroblastoma of sympathetic chain origin. (Figs. 3 & 4).

Treatment

On 3rd March 1956, Suprapubic cystostomy was carried out because of retention of urine due to paralysis of bladder.

On 5th March 1956, a decompression laminectomy was carried out. The mass was found to be infiltrating the muscles of the back on the right side with some extension to the left, through and in between them, and had infiltrated the laminae of the 3rd and 4th lumbar vertebrae and also the body of the 3rd. It had also infiltrated the dural sheath opposite the 3rd and 4th lumbar vertebrae. The mass was found to be avascular and of a light yellow colour which appeared more like myxomatous tissue. As much of the growth

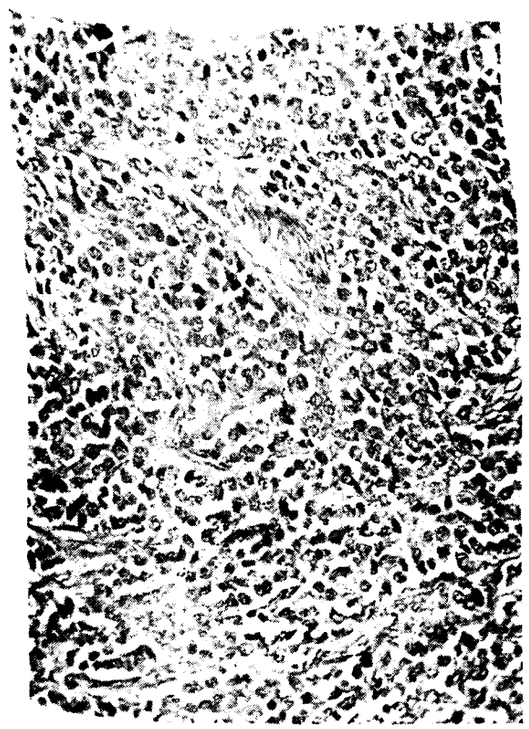

Fig. 3

Photomicrograph of the tumour. (X 240)

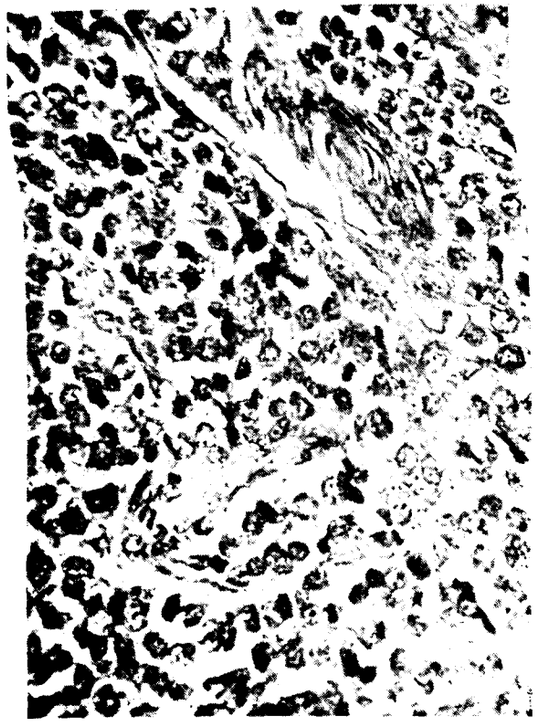

Fig. 4

Photomicrograph of the tumour. (X 480)

as possible was removed including the laminae of the 3rd and 4th lumbar vertebrae, to decompress the spinal cord. Wound was closed in layers with catgut, while skin was closed with No. 10 cotton thread.

On 7th March 1956, excision of the mass per abdomen was carried out by a transverse incision about 6" long. The growth was found to be arising from the right side of the bodies of lumbar vertebrae, pushing in front the psoas muscle, right kidney and ureter, inferior vena cava, and posterior parietal peritoneum. Peritoneum was incised between the psoas muscle and right ureter, and on lifting up the growth it was seen to be extending laterally to a distance of 4" from the midline. The growth was removed from all sides, intact, except from the muscles of the posterior abdominal wall, from which it had to be cut off. The mass was found to be capsulated and knobbly, finely granular on cut surface and firm in consistency.

Patient was then given deep X-ray therapy from second post-operative day onwards, with daily dosage of 200 r, till a total dose of 1,600 r. was given. Irradiation was focussed at the site of operation and remaining tumour mass, alternately from in front and from behind.

Post-operative course was uneventful and the wound healed well, except, for an attack of diarrhoea on 18th March 1956 probably as a result of cross-infection. This was controlled with appropriate doses of sulphaguanudine and bismuth kaolin mixture.

Further neurological examination done on 21st April 1956 revealed no improvement.

The child's condition continued *statusquo* till 2nd June 1956. Another course of deep X-ray therapy was given from 2nd June 1956 to 3rd July 1956. The dose given was 270 r daily for 20 sittings making a total dose of 5,400 r.

The child had fever for 2 days from 26th July 1956. X-ray of chest was taken and was found to be normal. The child was put on a course of Tetracycline which brought down the temperature.

On 3rd August 1956 the patient was discharged at request from the hospital with practically no further improvement. He carried the suprapubic tube with him.

Follow-up

The child died about 1½ months after discharge from the hospital, at his native place. Hence his condition at the time of death could not be assessed.

Discussion

This tumour mainly occurs in infancy, about 80% of cases occurring before the 5th year, while the sex distribution is almost equal. The present case conforms very much with the literature on this point. However, it differs from a large number of other cases reported in the literature in many respects. Nearly 60% of cases when first seen, already have metastases. The important feature that is seen in the present case is its presenting complaint—the patient came with paraplegia associated with a deformity in lumbar spine, and a mass in the right loin. He gave a history of fall on two occasions, 8 months before the paraplegia was noticed. The commonest cause of deformity of spine and paraplegia in children in this country is tuberculosis of the spine. It is not surprising to note that such an error in clinical diagnosis was made at the other two hospitals where the patient was seen before. He was hence advised chemotherapeutic treatment and immobilisation of spine for the same. Similar mistakes in clinical diagnosis have occurred in the hands of others, as reported by Chandler and McCorie Smith. The former author has reported a case which was considered as paraplegia due to post-vaccinal encephalitis. This patient was later proved to have a tumour, arising from within the spinal canal. Similarly, the latter authors have reported a case of metastatic deposit from a neuroblastoma in the head of the femur, being treated as a case of tuberculosis of hip joint. Paraplegia is not a very common complication in the life history of the tumour, but the radiological evidence of sclerosis of the 3rd lumbar vertebral body, with intact intervertebral discs, the destruction of the laminae of the 3rd lumbar vetebra, and granular calcification in the soft tissue shadow to the right of the vertebral column along with the firmness of the mass in the right loin, led us to believe that we were dealing with a condition other than tuberculosis of the spine, probably of the nature of an infiltrating growth.

The usual life span of patients having the tumour is very short. Although, it may vary, depending upon the nature of the tumour, the stage of the disease at which it is diagnosed and treated, and the response of the tumour to the therapy used. The present case can be said to have lived for 16 months after the onset of earliest symptoms, namely fall on two occasions which may well be due to early paresis.

Since an early diagnosis is all important, only the knowledge of the existence of such a condition and the various modes of its presentation puts one in a better position to make an early diagnosis.

As in the cases of other anaplastic tumours, mistakes in histopathological diagnosis do occur in cases of neuroblastoma sympatheticum. Such errors have been recorded by Seaman and Eagleton. They have quoted Ackerman and Taylor, having reported the mistaken diagnosis of Lymphosarcoma, and Hayman and Sztramski G. A., who have reported the mistaken diagnosis of Lymphatic leukaemia. It is not difficult to understand the occurrence of such mistakes, since, cellular structure, and cellular pattern of premature and dedifferentiated cells is so much alike. It also depends upon the experience of the pathologist in the cytology of tumours.

It is difficult to speculate on the exact site of origin of the tumour, since, the tumour had so far advanced that it had destroyed the laminae of the 3rd and 4th lumbar vertebra. Possibly it arose from the abdominal sympathetic chain.

Summary

(1) Recent literature on Neuroblastoma Sympatheticum is reviewed along with recent development in its treatment.

(2) The developmental stages in the formation of sympathetic system and its relation to various types of tumours is shown.

(3) A case of Neuroblastoma Sympatheticum with infiltration of vertebra and intraspinal extension is presented.

(4) Various errors in clinical diagnosis and histopathological diagnosis are discussed.

(5) The possible origin of the tumour, is speculated on.

Acknowledgment

Our thanks are due to Dr. M. D. Desai, Superintendent of the Hospital for permission to publish the case report ; to Dr. M. S. Kanvinde, Dr. H. I. Jhala and Dr. M. V. Sirsat for pathological report and photomicrographs ; and other colleagues for assistance.

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COMPLETE RUPTURE OF THE ILEUM FOLLOWING ROUNDWORM OBSTRUCTION

BY

S. S. CHADA, BOMBAY

Roundworm (Ascaris lumbricoides) infestation is quite common in our country. In a few cases, it may give rise to surgical complications, *viz.*, intestinal obstruction or perforation. This case deserves special mention as there was a complete rupture of the ileum at the actual site of impaction of the roundworms in the lumen of the bowel.

Case Report

L.D., Hindu female child aged about 5 years was admitted in the Medical Department of the Municipal General Hospital, Sion, on 20th June 1957 at 8-55 p.m. for the following complaints : Fever—duration 5 days ; Absolute constipation with occasional vomiting—duration 2 days. An enema was given at home on 19th June 1957 but it did not produce any result. The child had passed two roundworms in a bout of vomiting. There was a previous history of passing roundworms in the stools.

On 21st June 1957 at 10-30 a.m. the child was referred to the Surgical Department. On examination, the child looked very toxic. The abdomen was distended ; there was generalised rigidity and signs of free fluid in the abdomen. Peristaltic sounds were absent. Rectal examination did not reveal any abnormality. Temp.—103° F. ; Pulse—160/min., feeble ; B.P. 75/30 mm. of Hg. A pre-operative diagnosis of peritonitis following roundworm perforation was considered feasible. As the child's general condition was low, she was given injections of Cortisone and Achromycin along with a transfusion of blood which included Nor-adrenaline.

At 12-30 p.m., on the same day, an exploratory laparotomy was performed through a lower midline incision. On opening the abdomen the ileum was found to be completely ruptured about 12" from the ileo-caecal valve. The proximal segment contained a big bolus of roundworms and was gangrenous for about 3" from the site of rupture. Two roundworms were lying free in the peritoneal cavity which contained about 200 c.c. of purulent oxudate. The distal segment showed irregularity of the margin at the site of rupture. The proximal segment including the bolus of the worms was resected about 6" from the site of rupture ; the margins of the distal segment were freshened and an end-to-end anastomosis was performed. The abdomen was then closed up.

After the operation the child's condition remained precarious and she expired about an hour after the operation.

Discussion

Ascaris lumbricoides is well-known as a source of surgical emergencies in the tropics. Impaction of masses of worms of this type in the small intestine gives rise to acute obstruction. Sometimes they may even be the starting point of an intussusception, volvulus or acute appendicitis. One important characteristic is their pronounced tendency to cause pressure necrosis of the gut wall. This may result in intestinal perforation or even eruption of a suture-line. In this case the bolus of the worms caused a pressure necrosis of the entire circumference of the ileum leading to a complete rupture.

Summary

(1) A case report of complete rupture of the ileum following roundworm obstruction is presented.

(2) The various surgical emergencies associated with roundworms are mentioned.

Acknowledgment

I wish to thank Dr. H. S. Chitnis, Surgeon and Dr. S. V. Joglekar, Superintendent, Municipal General Hospital, Sion, for permission to publish this case report.

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NON-PARASITIC MASSIVE SOLITARY CYST OF THE LIVER

BY

S. S. ANAND, P. N. CHUUTTANI AND N. L. CHITKARA, AMRITSAR

Non-parasitic cysts of the liver, generally multiple, are not common. They are often associated with congenital cystic disease of the kidney, or other organs and congenital anomalies like meningocele, spina-bifida, talipes are frequent. Ackman and Rhee[1] reported 11 cases of non-parasitic cysts of the liver in 6141 necropsies. Davis[2] reviewed 499 cases of non-parasitic liver cysts including one of his own : these were multiple in 241, the cyst was solitary and unilocular in 187 while in 20 it was solitary but *multilocular* : no mention was made of the remaining 51. The right lobe of the liver is much more frequently involved by the non-parasitic cysts than the left lobe. McCangham and Rassiener[3] in reviewing 80 such cases including two of their own, found 52 patients with cystic tumours in the right lobe, 11 in the left lobe and 2 in the middle : in 6 cases the entire liver was involved while in the remaining 9 cases the site was not recorded.

We have not been able to trace a previous record of this condition in India.

Case Report

B., 20, male, resident of Bhutan, was admitted to V.J. Hospital, Amritsar on 14th November 1953 complaining of attacks of pain in the right hypochondrium for 1½ years. The attacks used to come irregularly. Pain was at times severe and occasionally accompanied by vomiting : it always radiated towards the right infrascapular region. Later he started feeling some fullness in the right hypochondrium which went on spreading downwards and for the last 6 months his abdomen had enlarged enormously. He had also fever off and on. On physical examination the liver was greatly enlarged and protuberant extending below the umbilicus. The upper limit of liver dullness was at the fourth rib in the mid-clavicular line. There was a low grade anaemia and the liver function tests showed a serum-bilirubin of 2.2 mgm"„ thymol turbidity 3 units, thymol flocculation 3 , cephalin cholesterol flocculation [(after 24 hours), 2 (after 48 hours) and total proteins of 6.8 gms"„ with serum albumin 4.1 gms"„ and serum globulin 2.7 gms"„. Urine for *urobilinogen* was positive but there were no bile pigments. Wassermann reaction was negative. Casoni's test was negative. Clinically before serological tests, diagnosis seemed to rest between hepar lobatum or a hepatoma although one could muster reasons against both. Hydatid cyst was regarded unlikely on account of negative Casoni's test and lack of a cystic feel. To further elucidate the nature of hepatic enlargement, a liver biopsy was attempted on 30th November 1953 when 20 c.c. of clear watery fluid was withdrawn while injecting novocaine for local anaesthesia. The patient

Fig. 1
Section showing lining of the Cyst. X 90

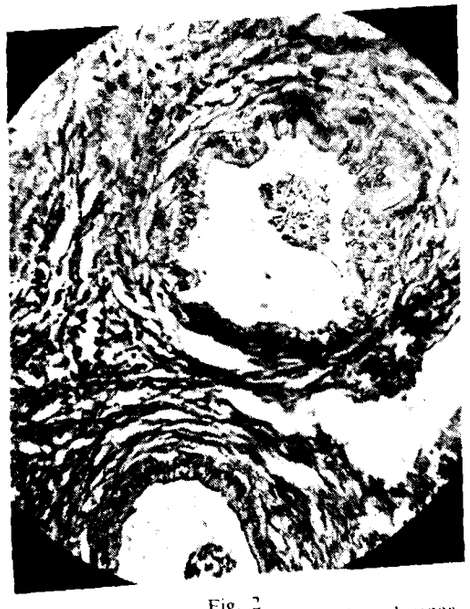

Fig. 2
Section showing dilated spaces lined by columnar epithelium. X 90

collapsed immediately afterwards and went into a state of severe shock reviving gradually on blood transfusions, intravenous glucose saline and antihistaminics. His condition was critical for about 12 hours. Examination of the clear watery fluid showed protein in traces and a few red blood cells and lymphocytes. No scolices or hooklets were seen. Free fluid in the abdominal cavity was detected on recovery from shock. Since we thought we had been led into the misadventure of rupturing a hydatid cyst, a laparotomy was done on 10th December 1953, when a smooth walled cyst could be felt on the postero-superior surface of right lobe of the liver. Loose pieces of cyst wall were present in the peritoneal cavity. Since the general condition was not good the cyst was aspirated and abdomen closed. Scrapings from pieces of cyst wall showed many leucocytes, epithelial cells and red blood cells. No scolices or hooklets could be seen. Wall of the cyst did not show laminated appearance or hooklets of hydatid cyst. Marsupalization of the cyst could not be achieved from the right paramedian incision, through which the exploration has been performed, as the cyst was lying very posterior.

A second operation was performed on 23rd February 1954 from behind with the idea of marsupalization. On opening the abdomen, it was found that the cyst had completely disappeared leaving a small knob of liver tissue near the anterior border. This piece was removed for histological examination : the liver cells showed pressure atrophy in certain areas. The wall of the cyst had at places no lining but cuboidal or flattened epithelium was seen elsewhere. In the fibrous tissue beneath the cyst wall lining were seen dilated spaces lined by columnar epithelium resembling dilated bile ducts.

Discussion

It is difficult to diagnose a cyst of the liver clinically as there is no clear cut picture. McCaugham and Rasiens in their review of 80 cases found very few cases diagnosed pre-operatively. In our case the clinical examination did not even suggest a cystic condition because of its deep situation.

The disease is most commonly seen in adult women in the ratio of 3 females to 1 male. Most authors agree that the cause of simple solitary cyst is some form of local obstruction in the biliary tract with a resultant retention type of cyst. However, there are divergent views on pathogenesis.

Mihalkowics et al[1] described the process as an inflammatory one (pericholangitic inflammation) resulting ultimately in dilatation of bile ducts. The inflammatory process during foetal life weakens the walls of the bile duct and these dilate never to recover their original size. Pye Smith[5] considered the process as a degenerative one, beginning as vacuolation of the hepatic cells, vaculoes ultimately fusing with each other. Sobourin[6] held that the cysts were neoplastic in nature : irritation led to the development of newly formed bile ducts which later fused and dilated forming larger cysts by a breakdown of septa. He labelled the condition as cavernous biliary angiomas. Others have held that the condition was cystic adenoma or cystic fibro-adenoma analogous to an ovarian cyst adenoma. Still[7] suggested a malformation of some of the cells of the hepatic diverticulum in the embryo. these cells going on to form cysts. Moschcowitz[8] described aberrant bile ducts in the fibrous tissue of the portal spaces of cystic livers which are not present in normal livers. Such aberrant dilated spaces lined by columnar epithelium were seen in the fibrous tissue surrounding the cyst in the present case (see plate).

Summary

A case of massive non-parasitic cyst of the liver in a young male has been described. Clinical examination did not even suggest a cystic condition possibly because of its deep situation. The views as to the pathogenesis of the condition have been summarised briefly.

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ORTHOPAEDIC SECTION

RENAL RICKETS

BY

K. S. GREWAL AND PARAMJIT SINGH GREWAL, AMRITSAR

The usual types of rickets are nutritional in etiology and are classified as foetal, infantile, due to steatorrhea, puerperal osteomalacia, hunger osteomalacia etc. and they are all amenable to usual doses of Vitamin D (1000 10,000 units daily) and diet containing phosphorus and calcium. But there is another group of rarefying diseases of bones in children and adolescents who for all practical purposes show similar changes in the bones but are not amenable to the above treatment. This latter type of rickets is becoming more prominent as the nutritional rickets is disappearing especially in the west. Occasional similar cases are seen in practice in our country and unless we are alive to this possibility we may ignore or mismanage such individuals.

These cases are classified as :

1. Vit. D. resistant rickets.
2. Renal rickets.

Vitamin D resistant rickets is still a matter of dispute as to its etiology. Dr. Dent maintains that it is a form of renal rickets. He believes that it is due to minor defect in renal tubules and it is the excessive excretion of Vitamin D rather than its utilisation or defective absorption which causes this type of rickets. Large doses of vitamin D cure such a condition but may not cure the other types of renal rickets.

Renal rickets may be due to defect in the filtering mechanism (glomerular) or in the reabsorbing mechanism (tubular). Absolute distinction between the two, however, is not always possible. The congenital cystic disease of the kidney, congenital hydro-ureter, and chronic interstitial nephritis usually show both glomerular and tubular defects. There is one fundamental biochemical fact however which stands out prominently. The glomerular defect always produces retention of phosphorus in the blood and the defect in the tubules on the other hand increases the excretion of phosphorus in urine due to lack of its reabsorption from the tubules. With this latter condition the reabsorption of aminoacids and glucose also suffer giving rise to glycosuria, amino-acidurea etc. The name of Fanconi-de Toni is attached to this latter syndrome. In phosphorus retention type of rickets, the hyper-phosphataemia stimulates the parathyroids and consequently fibrosis and decalcification of the bones results in addition to the widening of epiphyseal plates.

In the tubular deficiency rickets on the other hand phosphorus is depleted from the body and there is no stimulus for the parathyroid and consequently fibrosis of the bones does not occur. There is hypophosphataemia and vitamin D gets depleted and typical rickety changes are produced at the growing ends.

There is still another group of renal rickets in which the defect is in the tubules also but the defect is confined to inability of the tubules to manufacture ammonia to neutralise the acid urine and maintain acid-base equilibrium in the blood. This goes under the name of Butler Albright syndrome. In this condition due to failure of ammonia formation bases like calcium and sodium are depleted in urine causing chronic acidosis, hypophosphataemia and sometimes renal calcinosis.

As these types of rickets are not common we have been stimulated to report a case of renal rickets who shows typical changes in the bones both radiological and in the form of obvious deformities. The case fits into a classical group of renal rickets as his kidneys show defective secretion and retention of phosphorus.

Treatment

Vitamin D should be given in large doses in the beginning and then the dose may be reduced to a minimum maintenance level. Dosage should be regulated by plasma phosphate and calcium estimations.

In cases showing acidosis alkalies should be given in large doses. 5–20 Gms. daily of sodium bicarbonate may be required.

Case Report

C.S., aged 12, belonged to an agriculturist family of Amritsar District, was admitted to the hospital on 15th April 1957 complaining of deformities of lower limbs and vague pains all over the body of one and a half years' duration and feeling of giddiness of one and a half months' duration. The history dated back to three years when the patient developed diarrhoea accompanied by mild continuous discomfort in the abdomen. He used to pass ten to fifteen loose motions daily, occasionally containing mucus but no blood. The diarrhoea lasted about a year and a half. Then he developed pains all over the body which were increased in the lower part of the trunk and lower limbs on walking. At the same time he noticed progressive forwarding bending of both tibiae. On rising in the morning he occasionally used to notice puffiness of the face especially the eyelids. During all this period he has become very weak and has lost good deal of weight, so much so, that for the last few months he has to use a stick for walking. For the last one and a half months he feels giddy on standing and walking.

Personal and family history : He is a vegetarian, diet consists chiefly of pulses and chapaties. Occasionally gets vegetable, consumes about 8 ounces of milk daily. Parents are alive and healthy. He is the eldest child in the family, has three sisters and one brother living, all healthy. Two younger brothers died in their infacy —one of pneumonia and the other of some prolonged fever. There is no history of similar disease in the family.

Fig. 1
Showing bowing of tibia and femur on both sides and genuvalgum.

On examination he presents a pale, sallow and earthy complexion, poorly built and nourished, looks anaemic. Dental hygiene is poor, teeth are yellowish with tartar deposit. Tongue is moist and clean. Heart and lungs —no abnormality, liver and spleen—not palpable. Small discrete glands are palpable in both posterior triangles, epitrochlear and superficial inguinal regions.

Muscular skeletal system : Patient feels difficulty in rising from a squatting posture, stands in an attitude of slight flexion at the hips and knees and there is increased lordosis of lumbar spine. There is outward bowing of both femora and forward bowing of both tibiae. There is genuvalgum of 5". Costochondral junctions are slightly prominent and skull appears large in size. (Fig. 1).

Laboratory investigations

Total Protein	:	6.2 per cent.
Albumin	:	3.9 per cent.
Globulin	:	2.3 per cent.
S.T.S.	:	Negative.
Serum calcium	:	9.6 mgm per cent.
Serum phosphorus	:	5.0 mgm per cent.
Acid phosphatase	:	0.8 B.U. per cent.
Alkaline phosphatase	:	16.1 B.U. per cent.
Blood urea	:	55 mgm per cent
Blood sugar	:	81 mgm per cent.

Fundus :

N.A.D. : No abnormality in cornea, no deposit of cystine. Crystals.

Blood :

R.B.C.	:	3.7 million per cmm.
Hb.	:	10.4 G per cent.
P.C.V.	:	36 per cent.
M.C.V.	:	97.3 C.
M.C.H.	:	28.1
M.C.H.C.	:	29 per cent.
B.S.R.	:	34 mm.

Urine :

Cystine	:	Present.
Volume in 24 hrs.	:	750 c.c.
Calcium	:	10.5 mgm per cent.
Urine calcium per 24 hours	:	78.5 mgm.
Sugar	:	Nil.
Reaction	:	Alkaline.
Albumin	:	Traces.
Miscroscopic	:	N.A.D.
Urine urea	:	600 mgm per cent.
9·4 29·4 1·5		28·5 18·6
68 70 mgm. 90 mgm.		71 60
Urea clearance test	:	16 c.c. of blood cleared of urea per minute(21.3 per cent of normal).
Intravenous Pyelogram	:	No function shown by either kidney up to 22 minutes.
B.P.	:	98.60 m.m./Hg.

Roentgenograms of different bones show changes of rickets. (Figs. 2-5).

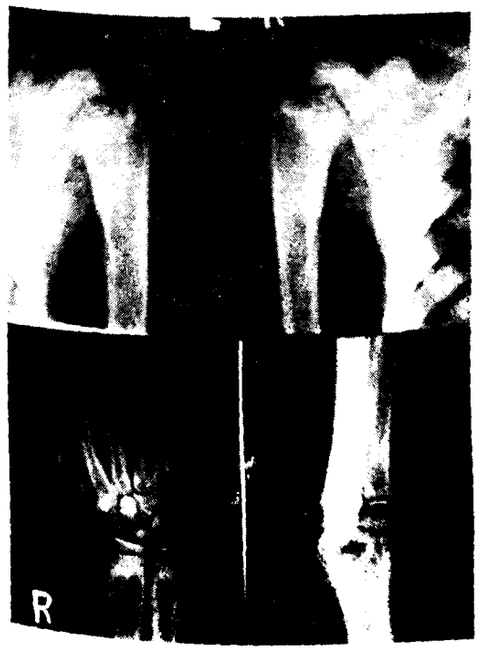

Fig. 2
Showing rickety changes in upper ends of humerus and at the wrist and elbow.

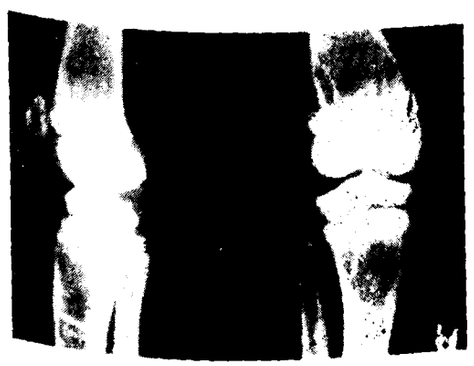

Fig. 3
Showing changes in the lower end of femur and upper end of tibia.

Treatment : Patient was put on massive doses of Vitamin D 50,000 units daily. Bone pains improved considerably. Surgical intervention for correction of deformities was considered inadvisable on account of the renal damage.

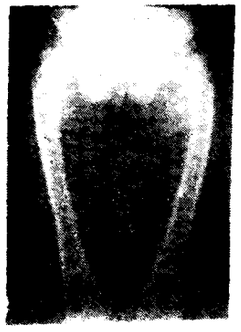

Fig. 4
Showing coxa vara and bowing of both femurs.

Fig. 5
Showing bowing of both tibiae and changes at their lower ends.

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A REPORT ON CASES OF LUMBAR DISC LESIONS TREATED DURING THE LAST FIVE YEARS

BY

V. JANARDANAN NAIR AND K. C. NAMBIAR, MADRAS

INTRODUCTION

The syndrome associated with a disc lesion has been a household word for centuries—sciatica. It seems reasonably certain that disc lesions are the commonest cause of low backache with sciatic pain. Charcot was the first to describe the spinal deformity and Brissot coined the term sciatic 'scoliosis.' Mixter and Barr in 1933 presented their historic paper suggesting that the so-called chondromatous tumours in the spinal canal previously described were in reality nuclear protrusions and they also advocated surgical removal as the best line of treatment. This was the beginning of the modern knowledge about the disc syndrome and their treatment. In the series given below we are presenting the last nine cases treated by us during the year 1956–57 in detail with a discussion on the best possible way of managing such cases as tried by us. During the past 5 years experiences with 30 and odd cases of Lumbar Disc Lesions treated by us, we have had some opportunity of coming to certain conclusions as to the ways for correct diagnosis and treatment.

Discussion

Anatomical facts : The discs have a triple function. They bind the vertebral bodies, they form an integral part of the intervertebral joints permitting movement between vertebrae, and transmit body weight. From a clinical point of view the relationship of the nerve roots to the discs should be well known to be able to interpret the signs and symptoms correctly. The adult disc consists of three parts. The cartilage plates enclosing it above and below, the nucleus pulposus and the annulus fibrosus. There is no clear line of demarcation between the annulus and nucleus and they merge one into the other with increasing age. Surgically the most important relationships of the lower lumbar discs are posterior, where they are in contact with the contents of the spinal canal and postero-laterally where they form one of the boundaries of the spinal foramina. The relations of the nerve roots are extremely important. In the extrathecal part of their intraspinal course, the nerves occupy an anterior position in the spinal canal and are in the most intimate contact with the posterior surface of one intervertebral disc.

Pathology : The actual pathology of a prolapsed disc is progressive degenerative change involving the nucleus pulposus and the

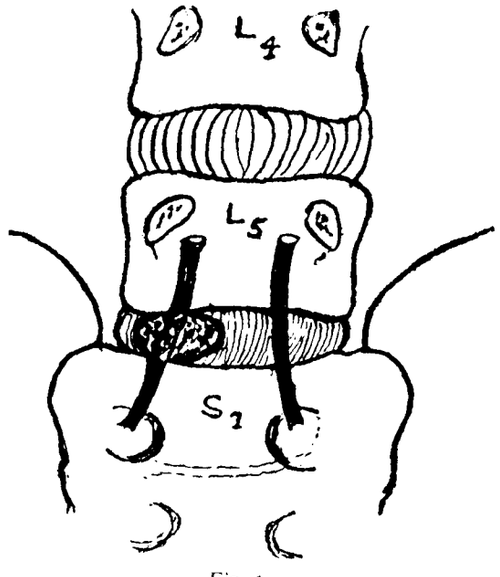

Fig. 1
(CASE NO. 1)

posterior part of the annulus fibrosus of lower lumbar disc. This degeneration is associated with a tendency to backward displacement of abnormal nuclear tissue through a posterior defect in the annulus, with arthritic change in the intervertebral joint. The commonest sites are the lower lumbar and the lower cervical regions. Trauma is considered to be an important factor in the causation of the prolapse. In our series six of the nine cases gave a definite history of some kind of injury direct or indirect to the spine. The changes in the lower lumbar discs during the disease are found to go through three stages.

First Stage : Consists of softening and disintegration of the nucleus pulposus : ligament becomes weak : no actual protrusion takes place.

Second Stage : Part or all of the nucleus gets protruded. The duration of this stage may vary greatly. The site of protrusion is usually to one or the other side of the midline and the central part rarely gives way : in our series all the positive cases were protrusions on one or the other side and only one was a protrusion on both sides. (The seventh case of the series) (see Fig. 3). A protruded disc may vary in size from a pea to that of a big marble and most commonly has a broad base and is rounded in shape. The various types of nuclear protrusions are (1) a bulge or protuberance—the first two cases in our series :

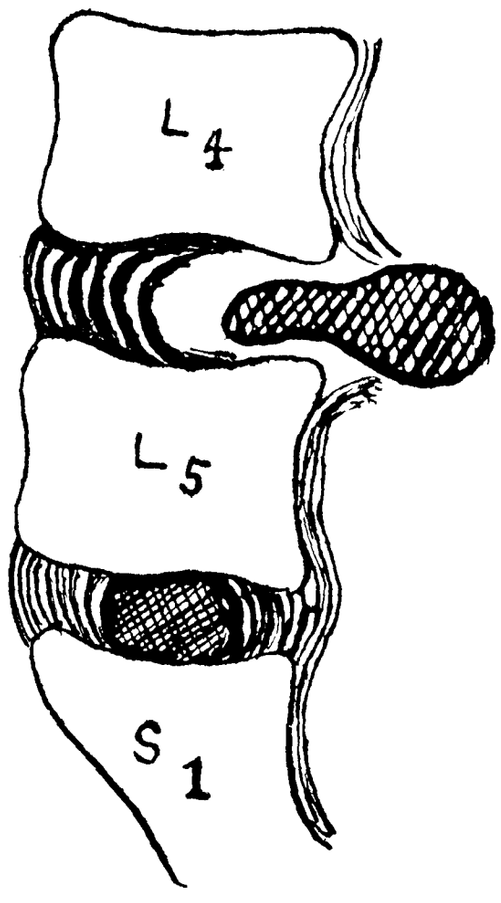

Fig. 2
(CASE NO. 5)

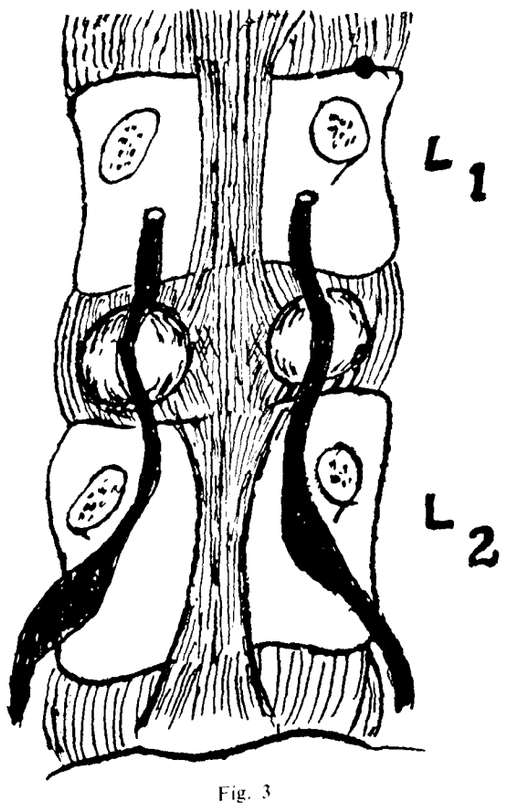

Fig. 3
(CASE NO. 7)

(2) sessile or pedunculated—the fourth and seventh cases in our series ; (3) the free sequestrum ; the fifth case in our series (see Fig. 2) ; (4) the dissecting protrusion—none in our series and (5) bilateral protrusion (see Fig. 3)—the seventh case in our series. The second stage may continue for varying periods, and the slightest movement of a sudden nature or manipulative treatment may aggravate the herniation.

The Third Stage : Constitutes the final stage of the disc lesion and is in some respects a process of repair—the disc gets fibrosed and calcified. The intervertebral space becomes progressively narrowed and the intervertebral joint is deranged, arthritic changes supervene and a firm fibrous ankylosis results in between the vertebral joints.

The secondary changes associated with disc lesions are—(1) the effects on the intrathecal roots of the cauda equina ; (2) effects on the cerebrospinal fluid ; (3) effects on the spinal theca ; (4) effects on extrathecal roots ; in our series none of the first two complications were observed : there was one case in which the protruded disc was adherent to the theca and in all the series, effects on the extrathecal roots were a marked feature and in two the protruded discs were firmly adherent to the concerned nerve roots. (see Fig. 1).

The discs most commonly affected are the fourth and fifth lumbar, the third lumbar disc is occasionally involved and lesions of the first and second lumbar discs are rare. In our later series reported here, of the seven cases operated on, two were in L4 space, one was in L3 space and one was in L5 space, one was in L1 space (very rare) and in two no definite protrusions were visible.

Symptomatology : The symptoms and signs in disc protrusion are quite characteristic and a correct diagnosis with an accurate localisation can be made by mere clinical examination alone. The clinical picture can be considered under three heads : (*a*) the history of the complaint as given by the patient ; (*b*) the physical signs ; (*c*) the results of certain investigations. There are few conditions where the patients' own history is so valuable in the final diagnosis. The classical picture is that of a man experiencing sudden and severe pain in the back, with a feeling of something snapping on the back while doing some kind of work either heavy or even of a trivial nature, like digging or getting up from the stooping posture or lifting a weight from the ground, and he remains locked in that stooped way, the slightest movement of the spine aggravating the back pain considerably. The pain usually shoots down the back of the leg, and coughing or straining increases the pain in the back as well as radiation down the leg which is the basis for Naffziger's sign. There may be anæsthesia, loss of tendon reflexes etc., according to the site and type of the disc protrusion. The posture of a patient in the throes of the pain is characteristic ; Lumbar spine is held rigid in a flattened position, whole trunk is tilted forward at the hips and some degree of scoliosis is seen on one or the other side. Straight leg raising test and Lasegue's test become positive. The extrathecal root has a normal range of movement when the straight leg is raised. With a disc protrusion the range is limited and root pressure increases and severe pain is experienced.

The causes for the production of symptoms and signs are many and varied. The low back symptoms are pain, muscle spasm, abnormalities of posture and disturbance of spinal movement. Pain in the low back may be due to pain arising from the nucleus itself or from the surrounding ligaments and joints. It may be a direct irritation of a root in its extrathecal course which may be responsible for referred pain from a posterior primary division of the concerned nerve root. Another variety of causation of low back pain may be due to a

nuclear sequestrum which becomes impacted between the posterior rims of vertebral bodies which produces a sudden, severe and stabbing pain in the back which may also as dramatically get relieved by disimpaction of the protruded disc. In the last stage of the disc lesion a type of low backache may be produced by the osteo-arthritic changes taking place in the intervertebral joints. The muscle spasm is of a protective nature to immobilise the affected joint. The abnormalities of posture associated with disc lesions are usually an obliteration of the normal lumbar lordosis and in some patients the formation of scoliosis. In all our patients low backache and deformities of the spine were marked features. The flattening of the normal lordosis is considered to be due to a joint derangement and the scoliosis is commonly associated with alterations in root tension. If the protrusion is para-radicular in site a lumbar scoliosis convex towards the lesion might reduce root tension while the converse would be true of a paracaudal protrusion. Scoliosis may also be due to joint derangement. Among the movements, forward flexion and extension are the most restricted. Symptoms referred to the legs are pain, sensory change, muscle weakness, muscle wasting and alteration in the knee and ankle jerks. Of all these, pain is the commonest of the symptoms and the others are relatively rare. In our recent series four cases had symptoms more than low back pain and shooting pain down the lower legs. Only one patient had—(Case No. 7) marked weakness of the lower limb, wasting of muscles and gross sensory change. These symptoms are due to the irritation of the concerned nerve root. Remission of symptoms is also a notable feature of disc lesions.

The special investigations that we carried out were a plain X-ray of the lumbar and sacral regions, routine lumbar punctures and Myodil Myelography. Of these, plain X-rays have revealed no abnormality in any of our cases except in one case where there was an associated sacralisation of the lumbar vertebra. Some cases showed osteo-arthritic changes. Myelography helped us in the localisation of the lesion in four of the recent cases reported on and in one case—Case No. 7—it showed an almost complete temporary block. This was the case with the bilateral protrusion. (see radiograph 3). Hence a negative Myelograph does not rule out the condition and it is not essential for a diagnosis.

Among the differential diagnosis the conditions to be thought of are : (1) Spondylolisthesis ; (2) Sacralised transverse process (3) Osteo-Arthritis of lumbar spine ; (4) Spondylitis Ankylopoetica ; (5) Intraspinal tumours ; (6) Pagets disease ; (7) Osteomyelitic Abscess and (8) Tubereulous Abscess. Each of these conditions have their own peculiar feature which can be easily distinguished.

The Treatment : Consists of (1) Conservative ; (2) Operative. In our recent series, seven cases were operated on and two cases

Fig. 4
Case No. 4
(Bifurcation of Dye at L_4 L_5)

were treated conservatively. All the cases operated on were given conservative treatment in the nature of bed rest and leg traction for nearly two weeks before an operation was undertaken. In all these cases symptoms and signs were not relieved by the conservative line of treatment and in three of the cases described above the lesions were so gross that nothing but an operative removal of the protruded disc would have cured the condition (Cases Nos. 4, 5 and 7) (Figs. 4, 5 and 6). The results in five of the seven cases operated on recorded above were excellent and the patients

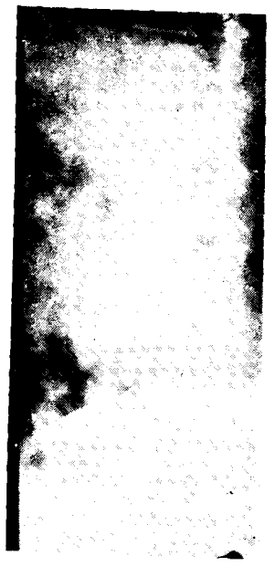

Fig. 5
Case No. 5
(Block at L_4 L_5)
(The Sequestrated Disc.)

had complete and gratifying cure. In the cases where definite protrusions of discs were not found, the results though not as dramatic and complete as in the other cases, they were definitely relieved of their symptoms and signs. In none of the above recorded cases were any bad side effects either due to the myelography or due to the operation, noticed. In our first 15 cases Lipiodol was used, and in the next

Fig. 6
Case No. 7
(Complete Block at L_4)

15 Myodil for myelography. Lipiodol used to produce mild side effects like fever but Myodil was found to be completely free from any side effect and we advocate Myodil for all Myelography. It is nearly more than 5 years in most of the cases since the operations were performed and on a follow-up of the cases none of them which were operated on have shown any recurrence of symptoms. As to the technique of operation some people advocate a hemilaminectomy and some others a complete laminectomy of 2 or 3 vertebrae. We have always done a complete laminectomy where good exposure of the field is achieved and any unsuspected protrusion in the adjacent spaces are also brought into view. In none of the thirty and odd cases that we have operated on have we done a post-operative spinal fusion and none of the cases has complained of any post-operative weakness in the low back. All of them have gone back to their normal occupations. In all our cases we have made our patients sit up on the 4th post-operative day and practise spinal exercises, and on the 6th day they have been asked to stand and walk short distances. We have used spinal anæsthesia in all cases except in those showing spinal block, and have found it extremely satisfactory, and even superior to general anæsthesia.

Summary

(1) Our experience with thirty cases of Lumbar Disc lesions have been presented.

(2) Case reports of the last nine cases in the period of 1956-57 are given in detail.

(3) A brief review of the features of the condition is given.

(4) The methods of diagnosis and line of treatment is discussed.

(5) The gratifying results of operative treatment are stressed.

(6) A rare case of disc protrusion in L_4 space is also reported.

Thanks are due to the members of the Neurology Clinic, Stanley Hospital, especially Dr. A. T. John, M.D., Physician and Dr. M. Natarajan, M.Ch. (Orth) F.R.C.S., Orthopaedic Surgeon for their valuable suggestions. Special mention must be made of Dr. B. Y. Pai and Dr. C. P. Joseph for their assistance.

We are thankful to Dr. C. Raghavachari, M.S., F.R.C.S., Dean, Stanley Hospital, for permitting us to report the cases from the hospital.

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Name (1)	Age (2)	Sex (3)	Complaint : presenting symptoms, gait ; deformity, duration (4)	Lasegue's test (5)	Naffziger's sign (6)	Deep reflexes (7)	Superficial reflexes (8)	Sensory defect (9)	Motor weakness (10)	Plain X-Ray (11)	Myelogram (12)	Treatment and result (13)
1. Athigounder— Adm. : 4- 4-1956 Dis. : 11- 5-1956	25	Male.	Pain in the low back of one year duration ; patient had a fall on his back ten years ago followed by the pain in the back ; one year ago again a fall on the back ; since then the pain in the back is continuous and it is shooting down in the back of the left leg and also in the left testis ; limping gait. Scoliosis towards the left in the lumbar region.	+	+	NAD	NAD	Nil	Nil	NAD	NAD	Operation on 17th April 1956 : lumbar laminectomy third pedical was narrowed and appeared to constrict the theca : disc protrusion seen in the fourth space left side causing pressure on the fourth root : protruded cartilage removed completely. Post-operativeperiod ; pain completely disappeared in the left leg and little pain in the back. Result- Good.
2. Raju— Adm. : 4 4-1956 Dis. : 28- 5-1956	48	Male.	Low backache, pain shooting down in the right thigh, duration three months. Sudden snap in the back while lifting a weight 3 months ago ; unable to walk afterwards due to the pain ; Scoliosis to the right side in the lumbar region ; Spine flexed in the lumbar region while walking.	+	+	Ankle jerk lost on the right side.	NAD	Light touch lost below the middle of right leg.	Slight weakness on the right leg.	NAD	NAD	Operation on 19th April 1956 : Lower lumbar laminectomy : disc protrusion third lumbar space right side ; it was completely removed ; post-operative period pain in the back and right lower limb completely disappeared but slight motor weakness in both lower limbs which completely disappeared in due course : Patient was discharged on 28th May 1956. Result Good
3. Kanakaraj— Adm. : 30- 3-1956 Dis. : 11- 6-1956	30	Male.	Neuralgic pain in the sacral region and back of left thigh and left leg of one month duration. Insidious onset ; no remissions ; left sided limp ; right side lumbar scoliosis spasm of the back muscles.	+	+	NAD.	NAD	Nil	Nil	NAD	NAD	Operation on 1st May 1956 : Lumbar laminectomy ; ligamentum flava was found to be thickened and there was unusual quantity of fatty material and areolar tissue around the theca there were a large number of veins present ; fifth lumbar space was cleaned of all fat. There was no obvious protrusion of disc : post-operative period : pain relieved : no motor or sensory defect : discharged on 11th June 1956. Result -Clinically satisfactory.
4. Rajkumar— Adm. : 29- 6-1956 Dis. : 25- 7-1956	30	Male.	Piercing and shooting pain, back of the left leg of two months' duration ; previous attacks twice before one and two years ago. Sudden onset by lifting a weight two months ago with low back pain and shooting pain in the back of the left leg ; limp on the left side ; is not able to stand erect ; lumbar scoliosis on the left side normal lumbar lordosis obliterated.	+	+	NAD.	NAD	Diminution of vibration sense left leg.	Slight motor weakness in the left lower limb.	Sacralisation in the lumbosacral region (see radiograph 1).	Bifurcation of the dye in the fourth space.	Conservative treatment. Recumbancy and left leg traction was given for one week ; repeated morphia injections were necessary as the pain was so severe. Operation Lower lumbar laminectomy ; ligamentum flava between the fifth lumbar and first sacral was found to be half an inch thick. Intervertebral space between L-V and S-I narrowed ; the fifth lumbar root on the left side was found to be thickened and firmly adherent to an underlying mass. This was found to be a nodular protrusion of nucleus pulposus which was completely removed. Post-operative period ; completely relieved the back pain and the shooting pain in the leg ; operation on 9th July 1956. Discharged on 25th July 1956. Result- Very good.

Name (1)	Age (2)	Sex (3)	Complaint ; presenting symptoms, gait ; deformity, duration (4)	Lasegue's test (5)	Naffzigers' sign (6)	Deep reflexes (7)	Superficial reflexes (8)	Sensory defect (9)	Motor weakness (10)	Plain X-Ray (11)	Myelogram (12)	Treatment and result (13)
5. Chandrasekarachari— Adm. : 1- 9-1956 Dis. : 7-10-1956	50	Male.	Pain in the low back and shooting pain right lower leg of 10 days duration ; is not able to walk erect ; severe spasm of back muscles ; lumbar scoliosis present and lordosis absent ; had similar pain two years ago on the left leg ; got relieved after some kind of treatment.	+	+	NAD.	NAD	Nil	NAD	NAD	Partial hold up of dye in the region of L-4, L-5 space. (see radiograph 2).	Operation on 20th September 1956 : lower lumbar laminectomy : constriction of theca between L-4 and L-5 seen ; fifth right side root was adherent to the protruded disc which was visible. It was of one C'meter diameter lying almost extruded and easily removed. Post-operative period—Completely relieved of the backache and the shooting pain in the leg ; the flexion deformity and scoliosis completely disappeared. Result—Very good.
6. Kotilingam Adm. : 15- 2-1956 Dis. : 24- 3-1956	40	Male.	Pain in the right sacroiliac joint region and pain in the right leg since five months ; acute flexion deformity of spine due to spasm of muscles and scoliosis. Lumbar lordosis is absent ; limp on the right side.	+	+	NAD.	NAD	Nil	Nil	NAD	Hold up of dye at fourth space (partial).	Operation on 3rd March 1956 : Lower lumbar laminectomy ; large quantity of fat was seen covering the theca posteriorly and also on all sides including the nerve roots ; on pulling long filaments seen in the fatty material. This was cleared by blood dissection ; right fifth root was thickened ; no definite disc protrusion seen. Post-operative period ; spinal deformities less marked ; pain in the back and leg relieved. Result—Good.
7. K. K. Nair Adm. : 2- 2-1957 Dis. : 2- 4-1957	50	Male.	Pain in the back after some strenuous work, radiating down the left leg of two months' duration ; feeling of numbness of left leg ; scoliosis to the side, lumbar lordosis obliterated ; back muscles stiff ; keeps the spine bent forwards on the hips ; left thigh and leg muscles appear wasted.	—	+	Knee jerks not elicited ankle jerks elicited on both sides.	Plantar reflex not elicited on left side.	Cotton wool lost pin-prick dulled upto umbilicus in front and gluteal fold behind vibration sense lost below the Antsuprliac spine on both sides.	Left leg weak.	NAD	Complete temporary block at L-1 level. (see radiograph 3).	Operation on 21st February 1957 : Upper lumbar laminectomy ; bilateral bulging disc protrusion adherent to the dura at L-1 level ; theca separated without damage ; discs scooped out on both sides ; left one was causing pressure on the nerve root also. Post-operative period ; Pain completely relieved ; loss of sensation and motor weakness recovered ; no spinal deformity. Result—Very good.
8. A. D. Nathan Adm. : 4- 8-1956 Dis. : 28- 8-1956	56	Male.	Pain in the back sacral region and the front of leg of 20 days' duration ; sudden onset felt something snapping in the low back on lifting a child ; patient not able to stand erect ; lumbar scoliosis present and lordosis absent.	—	+	Ankle and knee jerks exaggerated.	Plantar (?) extensor.	Nil	Left lower limb weak.	NAD	Filling defects in L-4, L-5 spaces in lateral views.	Conservative ; recumbancy in bed and analgesic drugs like Irgapyrin ; patient very much relieved. Sent home with the advice to come back if the pain becomes more severe. Discharged on 28th August 1956.
9. Chinnan Adm. : 14-11-1956 Dis. : 11-12-1956	20	Male.	Pain in the right sacroiliac region shooting down the right thigh and leg of duration nine months ; sudden onset while getting up from the bed when he felt a catch in the low back on right side ; severe and continuous for the last four months ; lumbar scoliosis present ; muscle spasm and flexion deformity of spine.	+	+	NAD.	NAD	Nil	Nil	NAD	NAD	Conservative : advised plaster jacket and recumbancy for four weeks : but patient was not willing for treatment. Discharged on 11th December 1956.

Routine investigations like B.P. urine analysis, Hb.%, ESR and VDRL tests were done on all these patients and were found to be normal. Routine lumbar punctures were done on all these patients and the CSF was sent for biochemical and bacteriological examinations and were found to be normal. All the discs removed were sent for microscopic examination and the reports were confirmatory.

Received for publication on 30-10-57

ASSOCIATION NOTES

THE XX ANNUAL CONFERENCE

The XX Annual Conference of the Association of Surgeons of India will be held at Visakhapatnam on the 27th, 28th, 29th and 30th December, 1958. The following Papers will be discussed :—

1. **Acquired Facial Defects**
   by Dr. M. Mukherji, Calcutta
2. **Hydronephrosis—Experimental and Clinical Studies**
   by Dr. B. N. Balakrishna Rao, Gwalior & Dr. A. E. deSa', Bombay
3. **Cysts of the Lung**
   (i) Hydatid Cyst of the Lung—
   by Dr. S. K. Sen, New Delhi
   (ii) Cystic Disease of the Lung—
   by Dr. G. S. Karai, Calcutta

Besides this, a few 'Short Papers' will also be read at the Conference. Members desirous of presenting short papers at the Conference are requested to inform the Hony. Secretary before the 15th of September, 1958 of their intention to do so and enclose a short precis of the paper at the same time. The time allotted for each paper is 20 minutes.

As the papers have to be circulated to a panel of referees who will decide regarding the acceptance or otherwise of the papers, members are specially requested to send in their complete papers (in triplicate) before the 15th of October, 1958. Papers received after the 15th October 1958 will not be accepted.

Subjects for the Main Scientific discussions at the XXIII Annual Conference in December, 1961

The 23rd Annual Conference of the Association of Surgeons of India will be held during December 1961. Three subjects for the main scientific discussions at the above Conference will have to be selected by the General Body at its annual meeting at Visakhapatnam in December this year. Members are requested to send in the titles of their subjects, if any, so as to reach the Hony. Secretary not later than 30th of November 1958.

Subjects for Discussion at future meetings

21st Meeting :

1. **Place of Vascular grafts in Obliterative Vascular diseases**
   by Dr. R. Nigam, Nagpur & Dr. V. V. DaSilva, Bombay
2. **Uses of Radio-active Isotopes in Surgery**
   by Dr. Miss P. Parvathi, Calcutta & Dr. S. R. Mukherjee, Calcutta

22nd Meeting :

1. **Reconstructive Surgery of the Biliary Tract**
   by Dr. S. J. Mehta, Bombay
2. **Clinical & Experimental Studies in Keloids**
   by Dr. K. K. Ghosh and J. L. Sinha.

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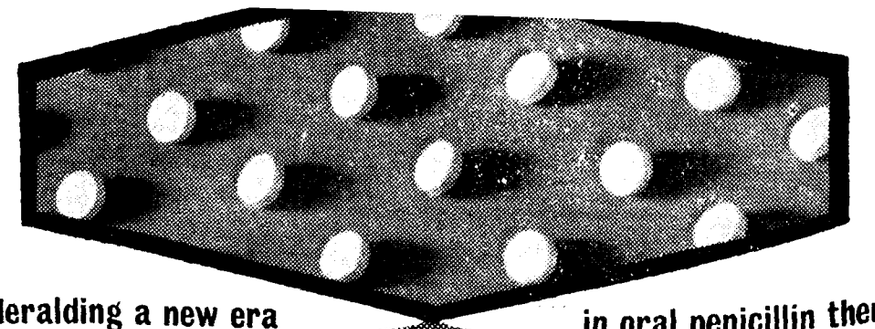

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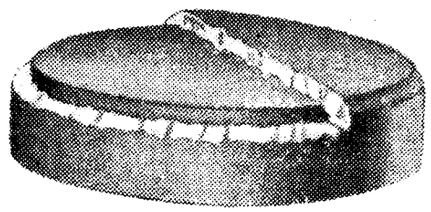

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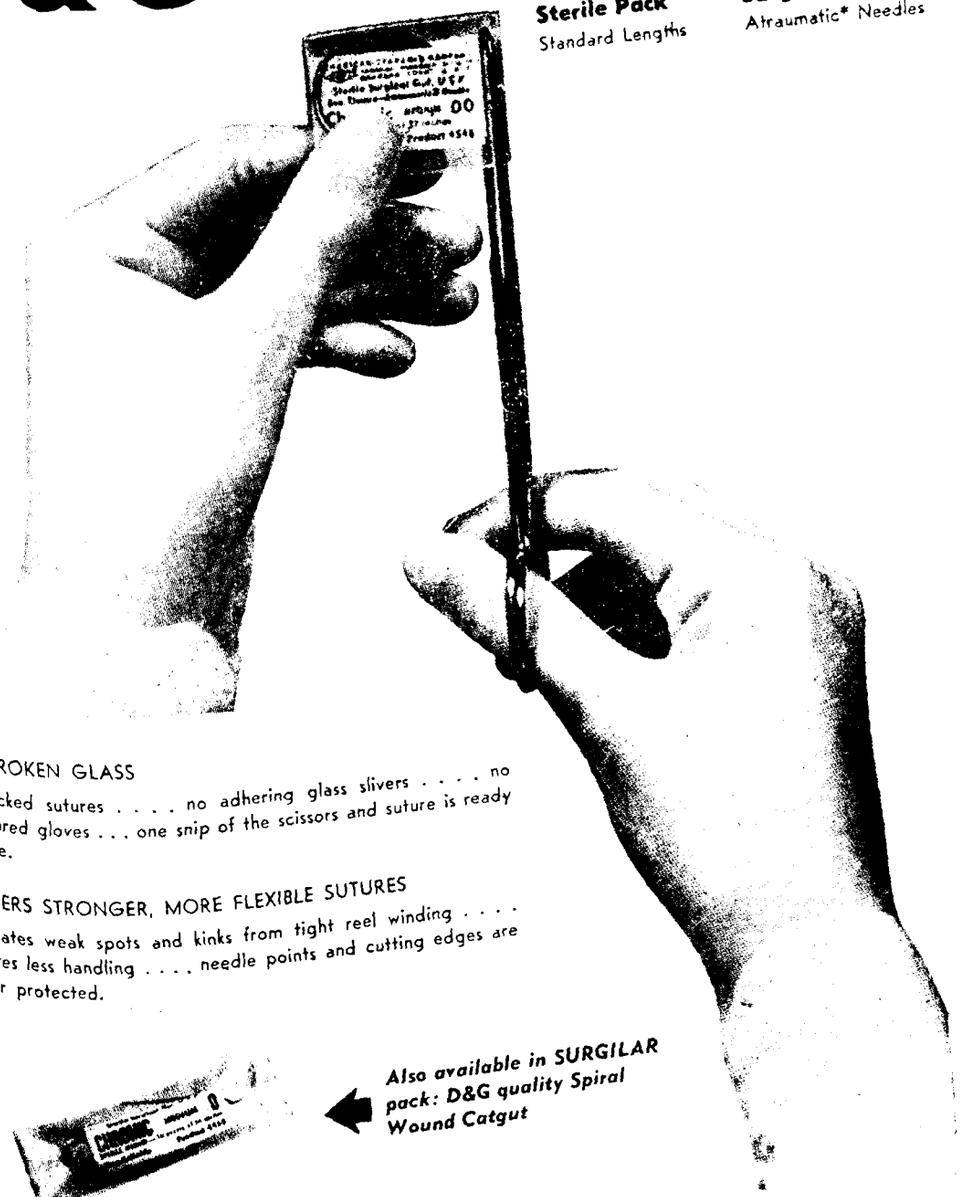

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THE
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PROBLEMS AND EXPERIENCES IN THE SURGICAL TREATMENT OF TETRALOGY OF FALLOT*

BY

REEVE H. BETTS AND N. GOPINATH, VELLORE (S. INDIA)

The surgical treatment of congenital cyanotic heart disease is based on the epochal work of Blalock and Taussig.[1] Approximately 75% of such cyanotic cardiac lesions are due to the combination of abnormalities known as the tetralogy of Fallot or a close variant of it. Numerous large series of cases have been reported from other countries, but there is a paucity of reports in the Indian literature of series from this country.[2] Although our group of surgically treated cases is not large, 47, and of comparative recent date, only seven prior to 1955, it seems worthwhile to record our experiences and some of the problems we have encountered in this modest experience.

Diagnosis

It is providential that the large majority of patients with congenital cyanotic heart disease have the tetralogy of Fallot and that in most instances these cases can be diagnosed without recourse to the more cumbersome and less readily available procedures of angiocardiography, or cardiac catheterization with pressure studies and blood gas analysis. There will always be a border-line group where all the diagnostic facilities will be necessary, but these more complicated tests are not indicated in the majority. Both angiocardiography and cardiac catheterization carry a small but definite risk in the severely cyanotic child. They should be used by all means when there are definite indications but not as strictly routine diagnostic aids.

A history of cyanosis from birth or early infancy, delayed development, limited exercise tolerance and squatting or other similar postures are most suggestive of the tetralogy of Fallot. Episodes of unconsciousness for a few moments, especially after exertion such as crying, are indicative of a severe degree of cerebral anoxia. A normal size heart on physical examination with usually a slight thrill and a systolic murmur maximal in the third or fourth intercostal space to the left of the sternum are to be expected. Examination of the blood reveals an increased hemoglobin content, sometimes over 20 grams, and the red blood cell count is elevated in some instances to 8 or 10 million per cubic millimeter in the more severe cases.

Radiologically the heart is not grossly enlarged, but the characteristic "boot shape" due to the enlarged right ventricle and diminished shadow of the pulmonary conus is found. The vascularity of the lung fields is also markedly decreased in most cases. The aortic knuckle is well seen as there is a relative enlargement of the aorta in these cases. In approximately 20% to 25% of patients there is a right aortic arch and a right descending aorta. It is imperative that the side on which the arch descends be determined by a barium swallow before operation as it influences the side on which the patient is operated upon.

A patient with tricuspid atresia and hypoplastic right ventricle may present much the same picture as a typical tetralogy of Fallot. The absence of any heart border to the right of the vertebral column in the anteroposterior chest X-ray and a left axis instead of a right axis deviation on the electrocardiogram indicate the presence of this condition. Differentiation should be made from the tetralogy of Fallot as the prognosis is not quite as good in tricuspid atresia although the same operative procedures are of benefit.

It is imperative that one differentiate the tetralogy of Fallot from pulmonary stenosis

* From the Department of Thoracic Surgery, Christian Medical College and Hospital, Vellore, South India.

without interventricular septal defect. There is usually no confusion unless the patient has an accompanying auricular septal defect with a reversed, or right to left, shunt. These patients have pulmonary stenosis on cardiac catheterization and may be mildly cyanotic. However, they are not benefited by a shunt-type of operation like the tetralogy of Fallot patients as this further increases the load of the overworked right ventricle without producing any benefit. Most of these so-called "pure" pulmonic stenotic cases where there is an intact inter-ventricular septum will have a pulmonary valvular stenosis and the post-stenotic dilatation of the pulmonary artery beyond the obstruction shows up in contrast to the small size of the pulmonary artery in the tetralogy of Fallot.

Other lesions with which the tetralogy of Fallot may be confused, such as the Eisenmenger's syndrome, auricular septal defect, ventricular septal defect or truncus arteriosus, are usually distinguished from the tetralogy of Fallot by the differences on physical examination and X-ray. When there is doubt, cardiac catheterization or angiocardiography or both may be necessary.

If a patient has a history of cyanosis from infancy, delayed development, squatting, an essentially normal-sized heart, a systolic thrill and murmur in the left third or fourth intercostal space and a "coeur en sabot" shaped heart on X-ray with normal or diminished pulmonary vascularity and the electrocardiogram shows right axis deviation, it is inadvisable, in our opinion, to carry out either a cardiac catheterization or an angiocardiography. When some of these cardinal factors are absent or when other signs or symptoms are added, one may need to exhaust the various diagnostic facilities to substantiate the diagnosis. Usually we have found cardiac catheterization with blood gas analysis to be far more helpful than an angiocardiogram. The pressure readings in the right ventricle and the pulmonary artery are the best means of establishing a diagnosis of pulmonary stenosis. Sometimes the cardiac catheter can be passed into the overriding aorta, but more often it cannot and the diagnosis of ventricular septal defect is made on the absence of any signs of an auricular septal defect by the pressure readings and sometimes by the slight arterialization of the blood in the right ventricle. A right descending aorta is suggestive but not pathognomonic of the tetralogy of Fallot rather than a "pure" pulmonic stenosis with auricular septal defect. It is found in approximately 20% of the tetralogy cases (found in six of our cases) but only rarely in the so-called pure case of pulmonary stenosis.

Treatment

Although an occasional case of tetralogy of Fallot will compensate and be able to lead a fairly normal life over a reasonable life span, most of them are apt to become worse as growth and activity add to the demand on the handicapped right ventricle. It is our opinion that most all cases of tetralogy of Fallot should be helped surgically. When symptoms are more severe and especially if there are periods of anoxic syncope, operation should not be delayed. The ages of our operative cases have varied from two months to 22 years (Table I and fig. 1). In those instances where there was no dangerous increase in red blood cell count or hemoglobin concentration and the patient was under four years of age, we have had the patient return every six months for examination, with operation being delayed until they have reached a more suitable age. From four to ten would seem to be the optimum age for surgical treatment.

TABLE-I

SHUNT OPERATIONS

	NO	FATALITIES	MORTALITY
PATIENTS LESS THAN 3 YRS	6	3	50%
PATIENTS 3 YRS AND OVER	41	3	73%

FIG. 1

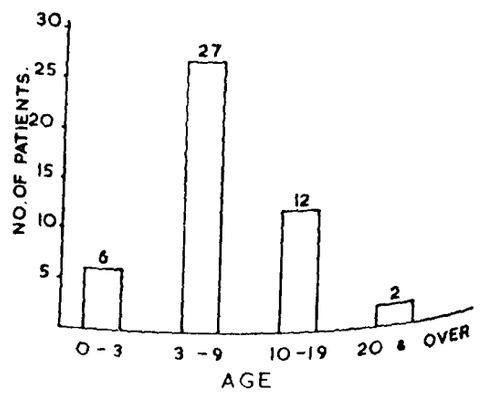

Fig. 1

This chart demonstrates the number of cases in each age group who had shunt operations. The six patients operated on under the age of three were severe cases that we thought could not safely be allowed to go on to a more suitable age.

We have a strong preference for the Blalock-Taussig type of surgical procedure. However, we prefer to go in on the side of the aortic arch rather than the opposite as advocated by Blalock in order that a Potts-Smith aorto-pulmonary artery anastomosis can be done should the subclavian-pulmonary artery anastomosis not be feasible. Our early experience was with the Potts type of operation, but the risk of making the anastomosis too large and causing congestive heart failure is a real one and we now prefer the Blalock operation where the size of the anastomosis is limited to that of the subclavian artery.

Our first four operations were the Potts-Smith type and since then we have used this procedure in three other instances, one being a child of two years where the aortic arch seemed placed very high and the subclavian not long enough for a Blalock. In the other two instances it was thought that the subclavian was too small for an adequate shunt. In one of these the aorto-pulmonary shunt which was constructed was made too large and the patient developed congestive heart failure soon after discharge and later died as will be mentioned below. Although we do not think that the type of operation was at all responsible, the two operative deaths we have had since 1953 both followed a Potts type of operation. We would still use the Potts procedure if confronted with a subclavian artery too small for adequate anastomosis or an arch of the aorta abnormally high, making a Blalock operation impractical.

In most of our earlier cases of the Blalock operation we used the subclavian artery after it had given off its first group of branches as we thought that the angle between the subclavian and aorta would be so acute as to prevent proper functioning if we divided the subclavian proximal to its divisions. This appears to be an erroneous supposition in most instances and we now try to use the subclavian before it branches, whenever possible. The aorta and its main branches are usually comparatively enlarged in a tetralogy of Fallot and hence the main subclavian is of good size and will give an adequate anastomotic flow. In those cases where a division was used there has been improvement but not as striking as when the main subclavian before it divides was employed and a larger shunt established. We routinely mobilize the inferior pulmonary ligament to allow the lung to ride up in the hemithorax and it is kept in this position during the operation by placing sponges between the lung and the diaphragm. In some instances we have also sutured the perihilar tissues of the pulmonary hilum to the aortic adventitia to further relieve any tension on the anastomotic site.

We have had no experience in the direct attack on the pulmonary stenosis in the tetralogy of Fallot as advocated by Brock[3] and Bailey[1]. It would seem a quite logical procedure in those with valvular stenosis but we believe with Lillehei[5] that if one is to make a direct surgical attack on the stenosis one should close the ventricular septal defect at the same time. Such a procedure entails an [redacted] cardiotomy with a heart-lung machine o[redacted] type and the mortality in the best of ha[redacted] still far higher than in the shunt type of opera[redacted] If one relieves the stenosis only without closi[redacted] the ventricular septal defect, the patient may become worse due to the large left to right shunt that then develops after complete relief of the pulmonic obstruction. No doubt the direct approach will be the adopted procedure in the future, but at present we are content with the results in the shunt operation until the other procedures become standardized.

For the past two years we have been using a mild hypothermia in all our cyanotic heart disease cases. We believe that lowering the body temperature moderately diminishes the risk of cerebral anoxia without materially increasing the risk from cardiac irregularities. We put the patient on the hypothermia blanket as soon as anesthesia has been induced and by the time we are ready to do the actual anastomosis the rectal temperature, which is measured by a thermocouple, will have dropped to about 35 C (95 F). The tank is then warmed to 43 C (110 F) with the pump turned off and warm water is circulated through the blanket as soon as the anastomosis has been completed. Even with hot water running through the blanket the temperature will usually drift down another two or three degrees before it starts to rise. We like to keep the patient in the operating room until the temperature has started to come up or at least has stopped falling and is stabilized before returning the patient to the ward. With this mild degree of hypothermia we have had no cardiac irregularities. In some instances when the temperature was as low as 32 C (89 F) it has not been possible for the anesthetist to record the pulse or the blood pressure even though the cardiac action seemed quite good on direct inspection. This is a common finding in hypothermia and seems due to the intense peripheral vaso-constriction which involves

even the larger arteries when the blood in them is cooled sufficiently.

There has been one serious complication in our series that we have not seen reported elsewhere in the literature and that is the loss of the left arm from ligation of the subclavian artery distal to its primary branches. In several instances in the literature[4] reference is made to the advisability of dividing the subclavian before the branching takes place in order to preserve some collateral circulation in the arm. This is not always possible and still have sufficient length to do a good anastomosis. In one of our patients (G. 35446) a 15 year old boy, a Blalock procedure was carried out on the left side and it was deemed necessary to divide the subclavian beyond the first branches. He went through the operative procedure well, but his left arm remained cold and blue after operation and he was unable to move it. In spite of giving papaverine and keeping the arm cool by packing in ice, it was soon obvious that the arm was non-viable. By this time, about 48 hours after operation, he also had a rather marked lower-nephron nephrosis syndrome. It was deemed imperative that any toxic absorption from the arm be prevented. So a tight tourniquet was placed around the arm and after he recouped from the lower-nephron nephrosis the arm was amputated. He eventually made a fine recovery as far as his cardiovascular status is concerned and his exercise tolerance is greatly improved. He is badly handicapped however by the loss of this left arm, but fortunately is right-handed. We have not encountered any report in the literature of such a complication before and we have been informed by Taussig[6] that they have not encountered it in the large series of cases at the Johns Hopkins Hospital. In most every instance it is advisable to divide the subclavian before it branches and use the larger vessel for anastomosis. However, there are quite a number of patients in whom the anatomical arrangement is such that anastomosis is possible only by going beyond the divisions. In such instances care should be exercised to see that the circulation in the arm is encouraged to develop and the arm kept cool until circulation is adequate. Several other patients have had temporary pain or inability to move the arm well for a short time, but these have all improved rapidly after the first few days.

Variations

As mentioned above, tricuspid atresia with hypoplastic right ventricle and auricular septal defect has certain characteristics in common with the tetralogy of Fallot, but it can be differentiated. This is the one other type of congenital cyanotic lesion that is also benefited by a shunt-type of operation. We had one such patient who was correctly diagnosed preoperatively by our cardiologist, Dr. K. L. Vythilingam. The patient was operated on and has considerably improved but not as dramatically as most of the tetralogy of Fallot patients. This is in agreement with other reports.

Everyone recognises that a patient with one congenital anomaly is apt to have others. We have encountered two anomalies that are of interest, both associated with right descending aortas which have been found in six of our cases. In one instance (I-42132) a girl of 8 years was brought to us with a typical history of tetralogy of Fallot. This was substantiated by the findings at cardiac catheterization and on angiocardiography. There was difficulty in establishing the location of the descending aorta, but it was thought that she had a left descending arch. At operation a curious vascular anomaly was found. There was a right descending aorta instead of a left, but the left subclavian artery arose directly from the pulmonary artery through the patent ductus arteriosus. A segment of this artery was resected in order to force more blood into the pulmonary circuit. As the patient had a right descending aorta and this anomalous left subclavian artery, it was not possible to do any shunt-type of operation through the left chest. Unfortunately, definite recording of the blood pressure in both arms was not done preoperatively. (It is now routine!). The patient was clinically improved after operation and we plan to do a shunt-type of operation on the right side should it seem warranted in the future. This patient taught us several things and had resulted in a "check list" type of study chart for all congenital heart cases so that such possible findings will not be overlooked in future.

Another case (R. 38754) a ten year old boy was diagnosed as a case of tetralogy of Fallot and it was found preoperatively that he had a right descending aorta. Operation was done through the left chest intending to use the left subclavian after its origin from the left innominate. At operation there was no left innominate and the subclavian arose from the right descending aorta but came around to the left behind the esophagus from lower down. The vascular ring around the esophagus and trachea was completed by the obliterated ductus arteriosus which was markedly stretched out and attenuated. It was divided before the anastomosis was carried out. The patient was very markedly improved by the Blalock type of operation.

Associated Pulmonary Tuberculosis

In a locality where the incidence of pulmonary tuberculosis is comparatively high one would expect to encounter a number of cases where pulmonary tuberculosis and the tetralogy of Fallot co-exist. We have now had three such patients. In one (A. 43463) the diagnosis of tuberculosis was not made preoperatively although, in retrospect, one can see some abnormal shadows in the left upper zone in the preoperative X-ray of the chest. At operation a moderate tuberculous infiltration of the left upper lobe was found. The Blalock operation was completed and the patient had an uneventful convalescence with antituberculous chemotherapy being instituted in early post-operative period. In a second case the patient (G. 43151) a man of 19 years came to the hospital because of hemoptysis of recent origin. He was markedly cyanotic. Examination revealed moderately advanced tuberculosis of the right upper lobe with cavitation and further studies including cardiac catheterization showed the cardiac lesion to be a tetralogy of Fallot. As his exercise tolerence was fair in spite of rather intense cyanosis, we felt that that the tuberculous lesion should be treated first. After three months of chemotherapy the right upper lobe was resected. His postoperative course was a bit worrying for the first few days as he developed a partial hemiparesis and facial weakness with inability to talk for 24 to 36 hours. This was presumably due to a spasm or thrombosis of one of the cerebral vessels. With papaverine and other symptomatic treatment the lesion cleared completely. The patient has been advised to return in six months for a Blalock operation on the left side. A third patient (L. 45536) was a 24 year old girl who complained of hemoptysis. Cyanosis had been noted since early childhood and the first hemoptysis had been thirteen years previously at the age of 11. X-ray examination showed a moderately advanced bilateral pulmonary tuberculosis with cavitation. Investigation including cardiac catheterization demonstrated a tetralogy of Fallot. She is now taking chemotherapy and a decision will be reached later as to when the cardiac lesion should be operated upon.

Another recent cyanotic patient was found to have active bilateral pulmonary tuberculosis, but his cardiac lesion has been proved to be an auricular septal defect with a right to left shunt. He is now in a sanatorium being treated for the tuberculosis.

Results

It is only to be expected that the early results of an operative procedure will not be as good as the later ones as experience with the operation should improve the technic. Also in the early stages only the more desperate cases are selected for operation. This has been true in our experience as illustrated in fig. 2.

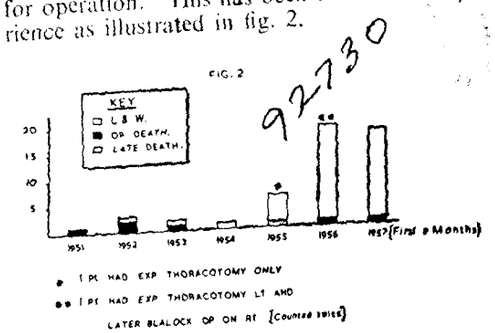

Fig. 2

This chart demonstrates the number of patients and indicates the operative and late mortality by years. It is only since the beginning of 1955 that any sizable number of patients have been treated. Of the 18 patients operated in the first nine months of 1957, as shown on the chart, one patient (referred to in the text) did not have a shunt operation but only a resection of an abnormal subclavian artery.

During the years 1951–1953 only six cases were operated upon. Three of these died within 48 hours of operation. A fourth lived just over two weeks and at post-mortem was found to have a complete transposition of the great vessels with other anomalies including a large porencephalic cyst. A fifth survived over a year although she had a hemiplegia after operation. She later died of subacute bacterial endocarditis. The other case, the only long-term survivor in this group, was operated on by Dr. Erik Husfeldt when visiting on a W.H.O. team. This very dark picture has improved materially in the latter period. In the last 41 cases who have had shunt operations there have been two operative deaths and one late death (Table 3). Post-mortem examination was obtained on both the operative fatalities. In one instance (V. 39612) it was found that the patient did not have a tetralogy of Fallot but an extremely marked infundibular pulmonic stenosis with an auricular septal defect and

Fig. 3
Postmortem photograph of patient S. 40308. One can see the extra valve placed beneath the aortic valve which must have markedly obstructed the outflow tract.

TABLE 2

SHUNT OPERATIONS

	NO OF PATIENTS	OP DEATHS	LATE DEATHS
POTTS OPERATION	7	5	2
BLALOCK OPERATION	40	1	0
TOTAL	47	6	2

TABLE 3

SHUNT OPERATIONS

	Blalock			Potts					
	OPERATIONS	FATALITIES		OPERATIONS	FATALITIES		TOTAL	TOTAL FATALITIES	
		EARLY	LATE		EARLY	LATE		EARLY	LATE
1951-1953	2	1	0	4	3	1	6	4	1
1954-1957 (Feb.)	38	0	0	3	2	1	41	2	1
TOTAL	40	1	0	7	5	2	47	6	2

must have had a right to left shunt to account for the marked degree of cyanosis. Although we knew that there were certain elements against the tetralogy of Fallot diagnosis, it was thought that the child had this lesion even after in angiocardiogram and cardiac catheterization. We believe that with further experience we would not make this same error in diagnosis again. The other operative death (S. 40368) was a two year old boy with evidence of marked right heart strain on the electrocardiogram. The patient died about 24 hours postoperative and the post-mortem examination revealed an additional valve cusp below the aortic valve (fig. 3) which quite obviously would balloon out and markedly obstruct the outflow tract to the aorta. We believe this was the cause of death in this patient.

The one late death in the last 41 cases was in a patient on whom we had done a Potts operation (P. 27491) because of a high placed aorta. On check up three months after operation she was found to have signs of congestive heart failure. This was undoubtedly due to the anastomosis being too large. Hospitalization was advised but not heeded and we have been informed that death took place not long after-wards. It is well recognised that the one big drawback in the Potts operation is the tendency to make the anastomosis too large and the heart goes into right-sided failure. In the Blalock operation the size of the anastomosis is limited by the size of the subclavian artery.

In most series of patients the mortality rate for patients in the younger age group is higher than for those that are older. This is mainly due to the fact that most of very young patients are done at an early age because of the severity of symptoms and therefore they are a more seriously ill group. Where possible everyone prefers that the patients be at least four years of age as the arteries are a little easier to handle when the patients are of this age or older. When the severity of symptoms is such that operation has to be done earlier, then one can expect a higher mortality rate. Gross[7] has reported that in the last three years of their series ending 1 January 1953, there were 135 patients with a total mortality of 11%. For patients under 3 years of age, however, the mortality was 30% and for those over three years of age it was 7.1%. This same finding has been reflected in our results (Table I) although our series is much smaller. We have had six who were operated upon under three years of age with three fatalities, a mortality rate of 50%, whereas in the three or over age group there are 41 with three deaths or a mortality rate of 7.3%. Whenever possible we prefer to wait until the patients are in the older age group, but when symptoms are severe, and especially in the presence of periods of anoxic syncope, operation should be done without delay. The greater part of the increased risk in these young patients is due to the severity of the lesion rather than to the age itself.

One patient had only an exploratory thoracotomy as he was found to have a valvular pulmonic stenosis and at that time, which was early in our experience, we thought it would be advisable to go in later and do a direct valvotomy. The operation was therefore terminated and the patient was advised to return at a later date, but has not done so.

Another patient (D. 29485) was operated on twice. When he was first explored through the right chest it was found that there was an abnormal branching of the vessels from the arch of the aorta. Both the left and the right carotid as well as the right subclavian came off a large innominate, but the branching of the right subclavian was high up in the apex of the chest and the subclavian could not be brought down for a subclavian pulmonary anastomosis on this side. As he had a left descending aorta nothing else could be done through the right thorax. He was again operated on (D. 31270) through the left chest and a Blalock procedure carried out without difficulty.

Of the 47 cases in this series we have done a Potts-Smith operation on 7 and Blalock-Taussig operation on 40 (Tables 2 and 3). Although we do not think that the type of operation was the decisive factor, there has only been one operative death and no late deaths in the group that had the Blalock operation. Thus the mortality rate is an acceptable 2.5%. None of our Potts type operative cases is alive at present, there being five operative deaths and two late deaths in the seven patients so operated upon. (This is another example of the fallacy of attaching too much significance to figures alone).

Summary

Our experience in surgical treatment of patients with the tetralogy of Fallot has been reported. Forty-seven shunt type operations were carried out on patients varying in age from under two months to twenty-two years.

During the period 1951 1953 inclusive, there were six operations, but only one of the patients still survives.

From 1954 to 1 October 1957 there have been 44 operations on 41 patients. One patient had only an exploratory thoracotomy and another had an exploratory thoracotomy on the right side and a Blalock operation at a later date on the left. A third had a division of a subclavian artery arising from the ductus arteriosus. Of the 41 patients having shunt operations there were three who had the Potts type and 38 the Blalock variety. The two operative deaths and the one late death in the group since 1954 were all following Potts operations although we think that the operative procedure was not responsible except in the one late death from congestive failure. The two operative deaths were due to an error in diagnosis in one instance and an abnormal additional valvular cusp in the second.

We believe that the moderate hypothermia (not below 32 C 89 F) used in the past two years has been a beneficial factor.

It is substantiated that in spite of all efforts and diagnostic tests it is sometimes most difficult to be absolutely positive about the diagnosis preoperatively, especially in the younger age group.

Our experience is like that of others in that patients who survive the immediate postoperative period continue to do well and are greatly improved by a systemic-pulmonary shunt operation. We believe that a direct attack on the pulmonary stenosis and the ventricular septal defect is not justified until improved methods have been developed.

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EXPERIENCES OF LEFT HEART CATHETERIZATION*

(A Preliminary Report)

BY

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The operation of mitral commissurotomy has passed through its pioneer phase and has emerged as a reasonably satisfactory procedure permitting direct surgical relief of mitral stenosis. One of the earliest principles governing the selection of patients for mitral commissurotomy was to exclude those with evidence of significant mitral insufficiency. The recognition of mitral insufficiency was based on clinical findings including the presence of an apical systolic murmur. As surgical experience increased it became obvious that the clinical estimation of the relative degree of stenosis versus insufficiency frequently did not correlate well with the operative findings. Often it was an humiliating experience for the clinician to have the surgeon assess the valvular defect of an occasional patient operated on for mitral stenosis as predominantly mitral insufficiency, or to have the valve of a patient, given a diagnosis of aortic stenosis, present little obstruction to the surgeons instruments. Experience thus emphasized the inadequacy of ordinary methods of physical examination and standard roentgenographic studies in the diagnosis of valvular defects. Right heart catheterization has been used to record the so-called " wedge " pressures, which are considered to reflect actual pressure relationships within the left atrium. Combinations of phono-cardiography, ballistocardiography and electrokymography have been used to provide data bearing on this problem. But all these procedures are not sufficiently reliable. So a great need for an objective method of evaluating the degree of mitral insufficiency in cases of mitral disease became obvious.

The direct pressure measurements in the left auricle thus seems to be a logical step in the preoperative investigation. The left auricular pressure curve may then be more significant in the determination of the degree of mitral insufficiency complicating the stenosis.

As a result, technics for puncture of the left atrium to obtain recordings of the pressure

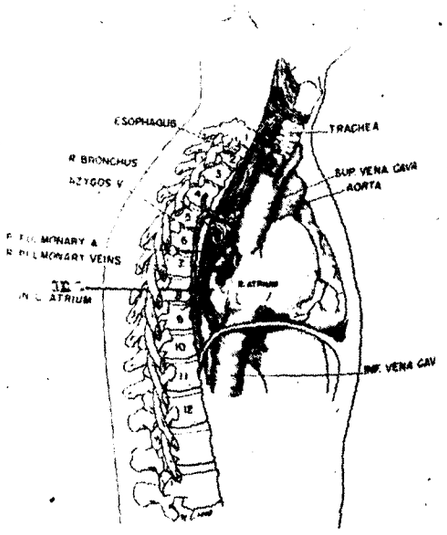

Fig. 1

Diagram showing the left atrial puncture needle in the left atrium, through the 8th intercostal space, which corresponds to the most prominent point of the left atrium in the lateral view.

were developed by the transbronchial route in 1952 by Facquet and associates[1] in France and subsequently used extensively by Allison and Linden[2] in England. In 1953 Bjork and co-workers[3] in Sweden developed the posterior percutaneous puncture technic. This technic has been modified and extensively applied by Fisher[4] and others in U.S.A.

Bjork, Fisher and others have extended the technic of left atrial puncture to a left heart catheterization by passing a small plastic catheter through the needle into the left atrium, and then advancing this catheter into the left ventricle and on occasion, through the ventricle into the aorta. The ability to record left ventricular pressure and thus to determine accurately

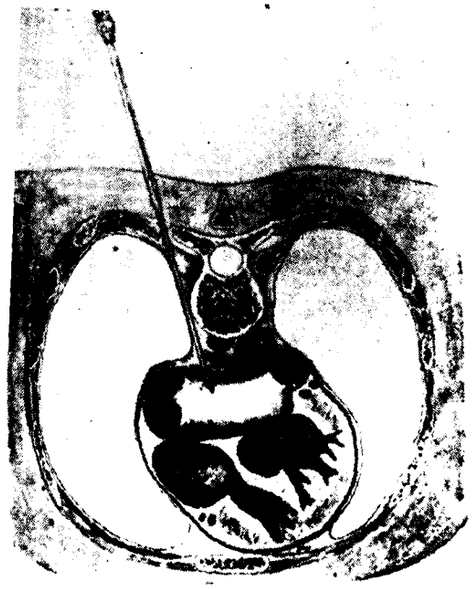

Fig. 2

Cross-section view showing the needle in the left atrium, introduced through the right paravertebral approach.

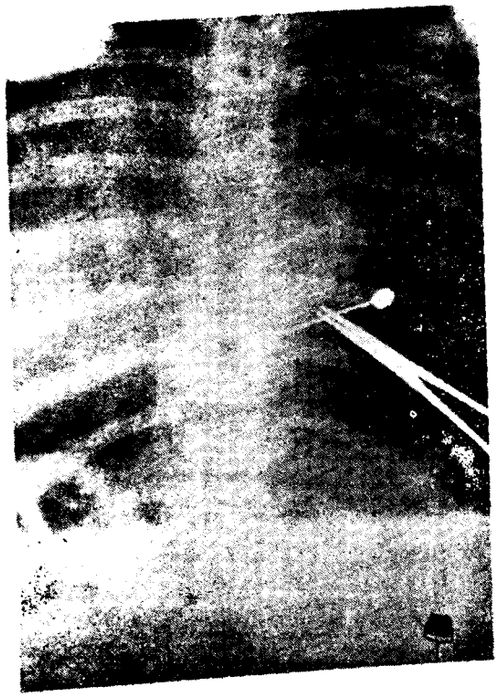

Fig. 3

Roentgenogram of case No. 6 in prone position as seen at flouroscopy with needle in the left atrium. Tip of haemostat indicates the point of skin entry on the right side.

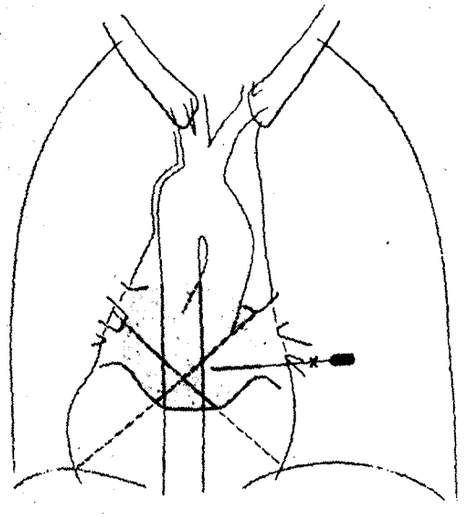

Fig. 4

Diagram indicating shaded area as the position of the left atrium on the heart shadow. The needle is directed from the point of skin entry towards the intersection of imaginary lines at the centre of the heart shadow.

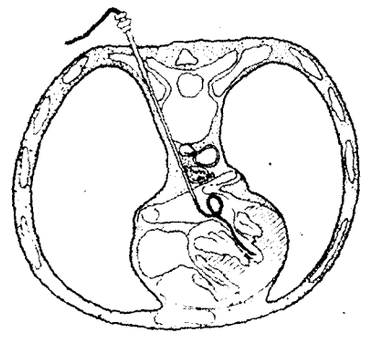

Fig. 5

Cross-section view showing the needle in the left atrium. The left heart catheter is threaded through the needle and then passed into the left ventricle through the mitral valve.

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Read at the 19th Annual Conference of the Association of Surgeons of India held at Nagpur, December, 1957.

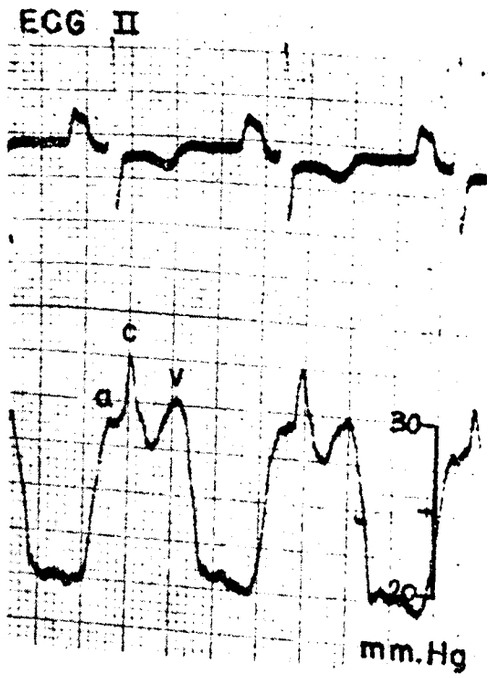

Fig. 8
Left atrial pressure recording in a heart with "tight" mitral stenosis and no insufficiency.

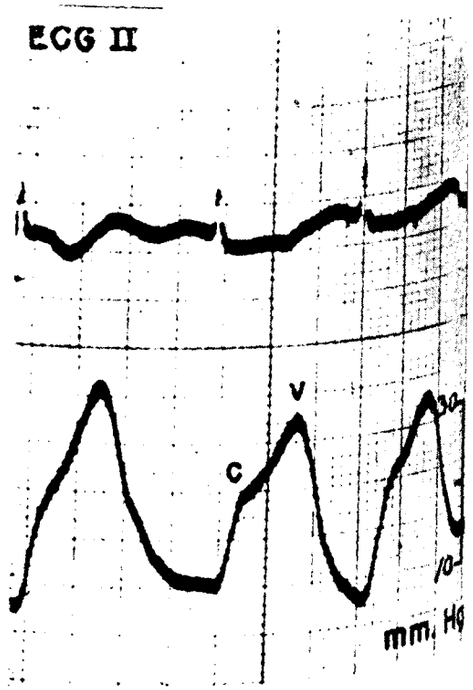

Fig. 9
Left atrial pressure recording in a heart with mitral stenosis and a severe degree of mitral insufficiency.

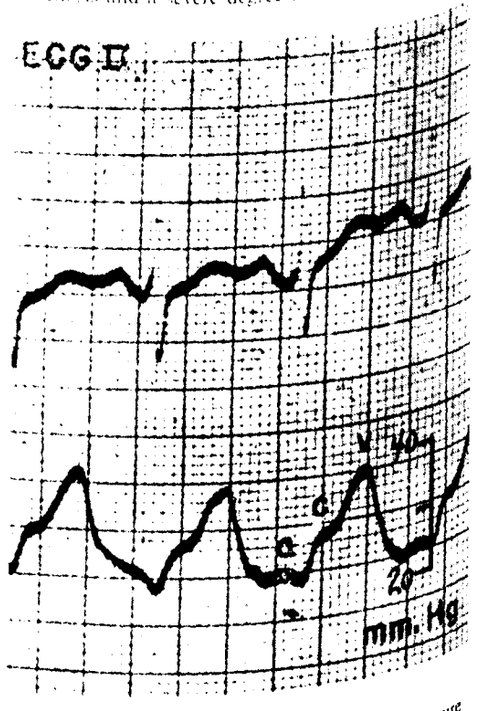

Fig. 10
Left atrial pressure recording in a heart with pure mitral insufficiency.
(Figs. 4, 7, 8, 9 and 10 are reproduced by permission of Dr. E. M. Kent and Editor, *Annals of Surgery*).

The left atrial pressure recording in the heart having mitral insufficiency alone is characterised by the following : (Fig. 10).

1. A mean pressure higher than 9 mm. of Hg.
2. An "a" wave having an amplitude of 3 mm. of Hg. or more.
3. A "c" wave having a tall upstroke of an amplitude greater than 5 mm. of Hg.
4. A "v" wave exceeding the level of the peak of the "c" wave by 5 mm. of Hg. or more. The down stroke of the "c" wave may be very short or absent.

A pressure gradient across the valve may be present but unlike mitral stenosis is found only in early diastole.

Complications

The complications and their frequency are found in Table I. There were only two major complications. In one patient, our first case, 1 foot of the catheter was cut off by the tip of the needle when the catheter was withdrawn before the needle was removed. The catheter was subsequently removed at operation from the left atrium.

In another patient, case No. 5 haemothorax of the right chest developed which was treated by aspiration and blood transfusion.

TABLE I

		Cases
Major		
1.	Catheter cut off ...	1
2.	Haemothorax Right ...	1
Minor		Cases
1.	Serosanguinous Pericardial Fluid	3
2.	Pleuritic Pain ...	3

Minor complications included sero-sanguinous pericardial fluid in 3 cases at operation. Three patients had pleuritic pain lasting for 24–36 hours. This is probably due to small amounts of blood in the pleura which could not be seen on flouroscopy.

Results

The results of this small series if cases is seen in Table 2.

Pure mitral insufficiency could be diagnosed easily in two cases (case 1 and 8). In case 1 it was confirmed at operation.

TABLE II

No.	Case	Age and Sex	Diagnosis: Clinical	Diagnosis: At Cath.	Diagnosis: Operative
1	Chandra 50501	13 Female	M.S. + M.I.	M.I. + M.S.	M.I.
2	Prabhakaran 50589	18 Male	M.S. + M.I. Aortic Lesion	M.S.	M.S.
3	Saraswathy 51325	35 Female	M.S. + M.I.	M.I. + M.S.	...
4	Kong 51749	36 Male	M.S. + M.I. ?	M.S.	M.S.
5	Arunachalam 53036	27 Male	M.S. + M.I. ?	M.S.	M.S.
6	Karthikeyan 51323	14 Male	M.S.	M.S.	M.S.
7	Suganamma 52611	21 Female	M.S. + M.I. ?	M.S.	M.I.
8	Amarjit 53491	17 Female	M.I.	M.I.	...
9	Kesavan 52351	12 Male	Aortic Stenosis + P.D.A.	Aortic stenosis	P.D.A. — Ligated Aortic Stenosis
10	Kettimathu 53382	16 Male	M.S. + M.I.?	M.S.	M.S.
11	Venkataraman 53949	36 Male	M.S. + Aortic Stenosis	M.S. + Aortic Stenosis	...

M.S. — Mitral Stenosis. M.I. — Mitral Insufficiency

In 5 cases (2, 3, 5, 6 and 10) we were able to diagnose pure mitral stenosis, though each had strong suspicion of mitral regurgitation. Case 2 has also a suspected aortic lesion. These cases would not have derived the benefit of surgery, had we not done left heart catheterization.

In one case (No. 4) we were able to diagnose mitral insufficiency to be the predominant lesion. This has not been confirmed at surgery.

There was only one case (No. 7) in which we were not able to diagnose mitral insufficiency, probably due to low mean atrial pressures. One case of aortic stenosis was diagnosed by retrograde left heart catheterization.

A few cases are presented for illustration.

CASE REPORTS

1. Mitral Stenosis (Case 10)

53382, a 16 year old male was admitted from the out-patient department as a case of mitral stenosis. He gave a history of rheumatic fever 3 years previously. The patient was having progressive disability to the extent that he was unable to climb one flight of stairs.

Physical examination revealed an enlarged heart. The apex beat was in the 5th left intercostal space. There was a distolic thrill ending in a slapping cardiac impulse.

In the mitral area a distolic murmur with presystolic accentuation ending in first sound was heard and a soft blowing systolic murmur was also present. In the pulmonary area there was a good blowing systolic murmur and a loud P_2. The liver was just palpable but not tender. The blood pressure was 90/60 Hg. The E.C.G. showed P Mitrale and Right ventricular hypertrophy and strain. On flouroscopy the heart was 55% of thoracic ratio and the pulmonary conus was prominent. This right ventricle was of normal size but the left atrium was enlarged.

A clinical diagnosis was made of mitral stenosis. This patient was selected for left heart catheterization because of the systolic murmur. The mean pressure in the left atrium was 50 mm. of Hg and the tracing pattern is typical of mitral stenosis (fig. 11). At mitral commissurotomy pure mitral stenosis was found.

2. Mitral Regurgitation (Case 8)

53491. This 17 year old female was admitted on 28 11 1957 with a diagnosis of rheumatic heart disease. She was sent to us by her physician for evaluation and surgery if indicated.

The patient had had rheumatic fever in 1950. She had been in right heart failure and was taking digoxin regularly and Diomax now and then for two years. Her exercise tolerance was limited to walking to the bathroom.

Physical examination revealed a precordial bulge. The apex beat was visible in the 6th left intercostal space, 2 cm. outside the mid-clavicular line. There was slight engorgement of the neck veins. In the mitral area a distolic thrill was palpable and a grade III systolic murmur was heard. The 2nd sound was followed by a long diastolic murmur. The pulmonary second sound was followed by a short

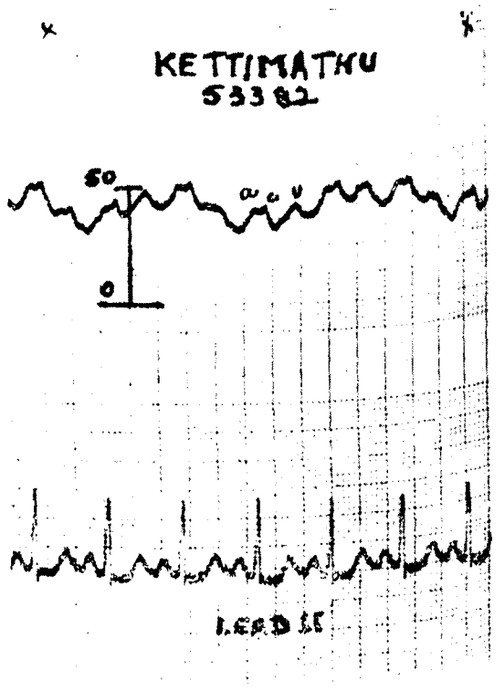

Fig. 11

Left atrial pressure recording in Case No. 10. Mitral Stenosis, showing a, c, v waves, and high mean atrial pressure.

diastolic murmur. There was a systolic murmur heard along the left sternal border and even conducted to the axilla. The liver was slightly enlarged. The blood pressure was 100/50 Hg. On flouroscopy the heart was 66% of the chest. The right ventricle was ++ and the left atrium ++ with systolic pulsations visible. The E.C.G. showed much hypertrophy and strain.

A clinical diagnosis of double mitral with predominant mitral regurgitation was made. This was confirmed at left heart catheterization (fig. 12). The tracing is characteristic of mitral regurgitation. The patient was advised to continue medical therapy.

There is another form of left heart catheterization which may be done in cases of isolated aortic lesions. In this a right heart cardiac catheter is passed retrogradely from the brachial artery into the left ventricle. The pressures are recorded in the same way as the catheter is withdrawn from the ventricle into the aorta. The pressure gradient across the aortic valve

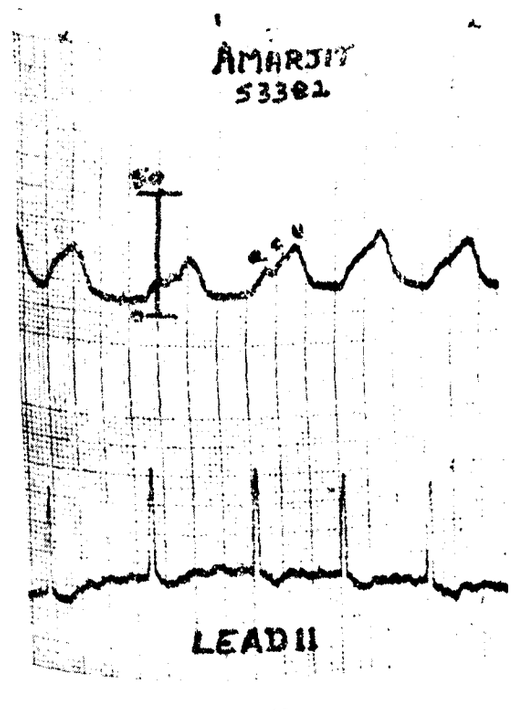

Fig. 12

Left atrial pressure recording of Case No. 8, mitral insufficiency showing immediate ascent of v wave following the c wave, due to the regurgitant pulse.

can thus be determined. This procedure was carried out in the following case which confirmed the diagnosis of aortic stenosis.

3. 52351 (Case 9) was an 11 years old boy admitted on 31 10 1957 with complaints of exertional dyspnoea and palpitation.

Physical examination revealed a marked thrill at the base of the heart. A systolic thrill could be felt over the most of the great vessels. The apex beat was in the 5th intercostal space. In the mitral area systolic murmur of grade IV with soft 2nd sound was audible. In the pulmonary area there was a continuous murmur with systolic and diastolic accentuation. In the aortic area a rough, grade IV systolic murmur was audible and well conducted to the neck. The blood pressure was 80/60 Hg. in the left arm and 100/60 Hg. in the right arm. The E.C.G. showed a left auricular dilatation and left ventricular hypertrophy. There was questionable right ventricular hypertrophy. A clinical diagnosis of patent ductus arteriosus with suspected aortic stenosis was made.

At right heart catheterization there was definite arterialisation of the pulmonary artery blood. The catheter could not be passed through the ductus.

A retrograde catheterization was done through the right brachial artery and the pressure tracing pattern (fig. 13) showed a definite pressure gradient across the aortic valve thus confirming the diagnosis of aortic stenosis. A retrograde aortogram done at the same time showed the presence of a ductus. The ductus was ligated without incident.

4. Aortic and Mitral stenosis (Case 11)

53949. 36 years old male was admitted with complaints of dyspnoea on exertion and precordial pain of four years duration. Patient could not walk more than two furlongs without getting dyspnoeic and getting precordial pain which lasted for about 3 minutes. There was no history of rheumatic fever.

Physical examination revealed an enlarged heart. There was diffuse pulsation felt at the apex. In the mitral area there was a faint diastolic murmur. There was a grade III systolic murmur heard in the aortic area. This murmur was conducted up in the neck. Pulse 92 per minute regular. Blood pressure 125/85.

X-rays of the chest showed a generalised cardiac enlargement with a prominent pulmonary conus. There was enlargement of left auricle.

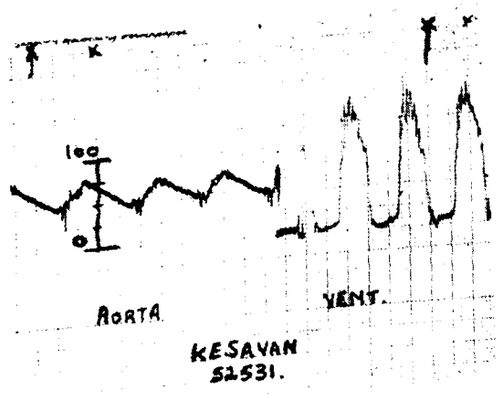

Fig. 13

Patient No. 9 aortic stenosis with P.D.A. Recording of aorta and left ventricle showing a marked systolic gradient as the catheter passes across the aortic valve (Retrograde).

A clinical diagnosis of aortic and mitral stenosis was made. Patient was selected for left heart catheterization to assess the presence of mitral stenosis and degree of aortic stenosis.

Left heart catheterization was done on 20–12–1957. The left atrial pressure tracing (fig. 14) shows elevated mean pressures and is characteristic of mitral stenosis. There is no evidence of mitral regurgitation. The left ventricle was entered successfully by the catheter. The pressures in the left ventricle (fig. 15) are of 200/0 mm. of Hg. An attempt was made to pass the catheter into the aorta through the aortic valve, but was unsuccessful. So a retrograde catheterization of the aorta was done through the left brachial artery. The pressure tracing of the aorta (fig. 16) shows pressure of 125/75 mm. of Hg.

Thus a diagnosis of severe aortic and mitral stenosis was confirmed by this left heart catheterization. Patient is awaiting surgery of both these lesions.

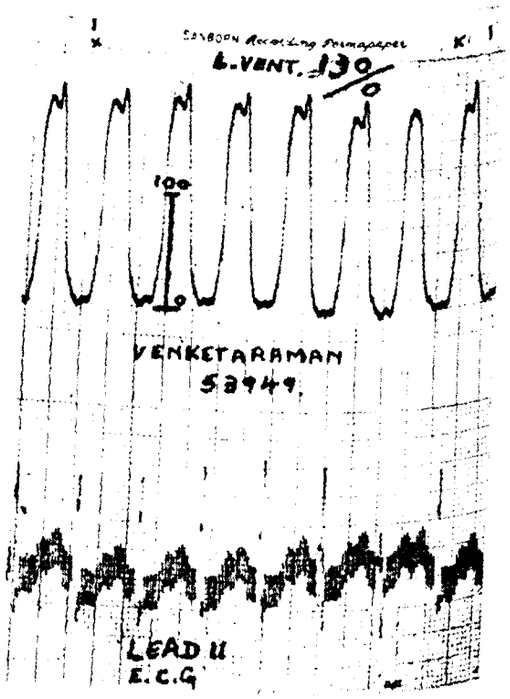

Fig. 15
Pressure recording of Case No. 11 in the left ventricle obtained by the left heart catheter.

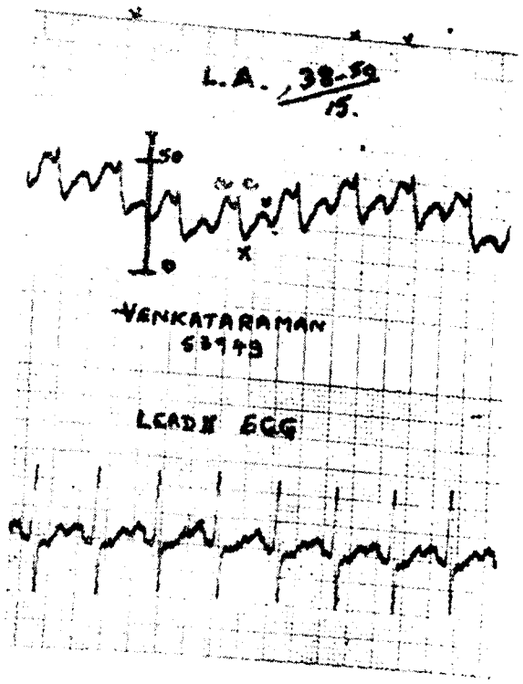

Fig. 14
Left atrial pressure recording of Case No. 11, Mitral stenosis. a, c, v waves can be seen. There is a negative x-wave before v wave. v-wave is lower than c-wave. Mean atrial pressures are elevated.

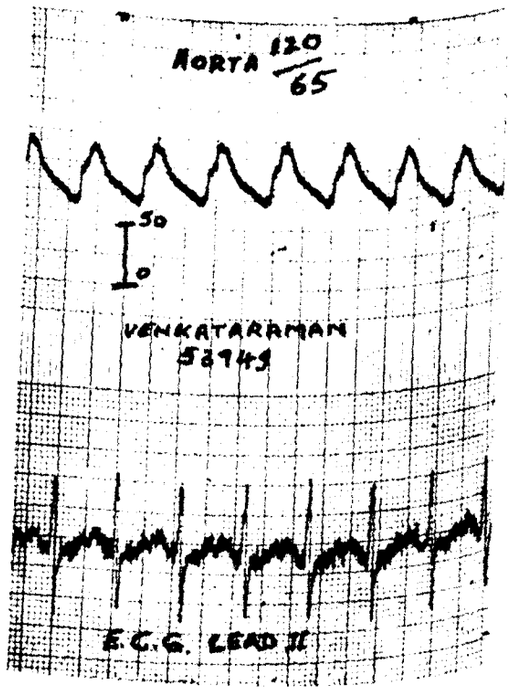

Fig. 16
Pressure recording of Case No. 11 in the ascending aorta just above the valves, obtained by retrograde left heart catheterization. There is marked systolic gradient across the aortic valve indicating aortic stenosis.

Discussion

The penalty is great for surgical exploration of a lesion which proves incapable of relief. Such patients die with disturbing frequency in the postoperative period, while on the other hand many patients can be given the benefit of surgery, if they have a remediable lesion. Accurate pre-operative diagnosis then becomes of paramount importance to prevent operation on patients with cardiac defects not amenable to operation, especially as there is no suitable operation for mitral regurgitation at present. For these reasons, the precision in diagnosis afforded by left heart catheterization is heartily welcomed. The greatest value of left heart catheterization, to the surgeon at least, seems at present to be in the accurate identification of mitral stenosis, mitral regurgitation and aortic stenosis.

In this series of cases, the comparisons show good agreement. In all cases, except one, where an operation has been performed, the diagnosis as to the relative severity of the multiple lesions and the decision as to the proper surgical approach were correctly influenced by the pressure interpretations. Although the test has been quite reliable, further experience is required before proper evaluation of diagnostic standards can be made. Some workers have extended this procedure in that they determine the rate of blood flow across the valves. Some have even measured the size of the valves by ingenious formulae. Others employ angiocardiography along with left heart catheterization. All these procedures stress one point and that is that in spite of the diagnostic acumen of clinical cardiologists, certain cases defy accurate categorisation. Therefore we believe that left heart catheterization can be of definite benefit in the diagnosis of certain type of cases and for lesions that are not clear cut clinically.

Summary

1. Eleven patients were subjected to left heart catheterization.

2. The technique of the procedure is described.

3. Eight of these patients were subjected to surgery and thus diagnostic interpretations were compared. Only one major disagreement occurred in these comparisons.

4. The series is too small to provide definite conclusions, but we feel that it is a good diagnostic aid.

5. No fatalities nor lasting sequelae resulted from the tests. There were two major complications.

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A NEW BLADDER MADE OF THE SIGMOID COLON

BY

NIKOLA DELEV, Sofia

The diversion of urine into the intestine is not a new procedure. The British surgeon John Simon was the first to divert urine into the intestine as early as 1951. The first implantation of ureters disconnected from the bladder was carried out by T. Smith in 1878 and consisted of the anastomosis of the mucous mucosis.

The diversion of urine into the intestine is indicated in the following cases : complete incontinence in ecstrophia vesicae, epispadias and hypospadias, inoperable vesico vaginal fistulae, irreparable blockage of the urethra, when the bladder has shrunk to the highest degree in consequence of cancer or tubercular or other inflammation of the bladder, in cases of cystectomy or as a palliative measure because of unbearable pain.

A difference should be made between methods aimed at diversion of the urine and methods in which continence is not preserved, the former presuppose an intact sphincter ani, while the latter are cases of pyelostomy and cystostomy. Ecstrophy or cancer of the bladder are the most frequent indications for diversion of the urine into the intestine. There are two possibilities :

(*a*) To implant the ureters straight into the intestine.

(*b*) To implant the ureters directly into a small separate part of the colon which is to replace the bladder.

Until recently it was believed that urine diversion had found its final solution in uretero-sigmoidostomy. This operation, which has lately been performed more frequently with greater facility and certainty, is nevertheless followed by some delayed and often fatal consequences. This has made the surgeons consider the problem of replacing the bladder with a new one, made of the large intestine, part of the skin or folds of the small intestine.

We performed cystectomy on 3 patients, who had constant incontinence due to a congenital anomaly of the bladder.

The patient N.Y., 17 years old, a housewife, has had the complaint from birth. The urine flowed constantly, soiling her body and clothes, causing itching, an unpleasant odour and eczema on the skin around the urinary orifice and the inside of the thighs. She has been treated many times, mostly in a conservative manner, without any improvement. A year before she had undergone an operation, an attempt being made to create an artificial urethra, but the operation was not successful.

The tests carried out on the patient showed light pleural adhesions at the base of the right lung, and a lightly expressed mitral vicium. The remaining tests gave normal results. The soft tissues in the pubic region were divided by a wide furrow which continued downward to the outer genitalia and separated the minor and major labia quite widely in their upper pole. The clitoris was lacking. The bladder orifice was visible under the pubic region ; urine flowed freely from it. The skin of the thighs, the seat and the vulva were inflamed, macerted and at places covered with suppurating papulae and crusts. The inflamed places were painful.

The blood picture and the sedimentation rate were normal.

Urine : alb.—negative, sugar—negative, relative weight—1,022, acid reaction, sediment—6 to 8 leucocytes. Blood urea 27 mgr. per cent. Venous pyelography and cystography—normal.

The pre-operative treatment lasted 5 days. The urine was drawn off through a permanent catheter and the inflamed skin was smeared with zinc paste. Four grams of sulphamides were administered daily to disinfect the intestine. Prior to the operation castor oil was administered to clear the intestine, followed by enemata.

The operative method had been first tried on corpses. Our purpose was to make a new bladder out of the sigmoid colon and to form a tube which would be the urethra of this new bladder. The sphincter ani was to close the new urethra.

To ensure the success of the operation, we carried it out in two stages :

(*a*) The first step was to make the new bladder and to bring its distal end parallel to the rectum intrasphincterally. In a short time it was to be cleaned and its function tested.

(*b*) The next step was to implant the ureters into the new bladder.

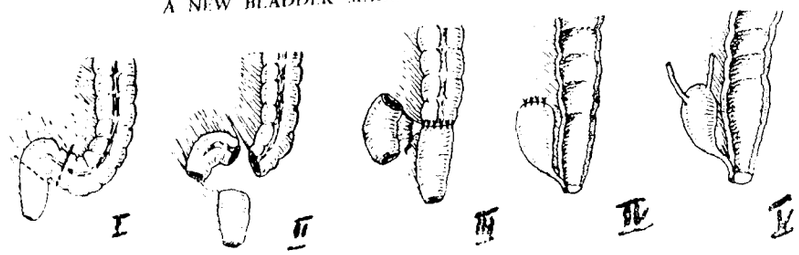

Fig. I

The first stage of the operation was performed on April 1, 1956. Posterior medial laparotomy was done under general anaesthesia with ether. The intestines were pushed up to the diaphragm and carefully isolated. The plica retrouterine was opened. A blunt tunnel was made between the ventral side of the rectum and the dorsal side of the vagina reaching as far as the perineum. A resection was made in the sigmoid section of the large intestine at the point of the greatest curvature. The length of the incised fold was about 25 cm. ; the mesenterium and the arteries were not cut. The incised fold was then separated and wrapped up in a gauze compress. (Figs. I-I & II). The passage of the large intestine was restored by joining it end to end. The oral end of the incised intestine fold was sutured with double stitches. The distal end was so reshaped that after narrowing it down it assumed the form of a pear (Fig. I-III & IV).

Thus shaped the new bladder was placed in the tunnel made for it. Part of the bladder stuck out of the tunnel, in the abdominal cavity and was peritonialised with the peritoneum incised in the region of the plica retrouterine. Several thick threads were tied to the end of the new urethra and left hanging. They were to be later used in pulling the urethra in the perineum region. Antibiotics were placed in the abdomen and the various layers were closed in succession.

The patient was placed in a gynaecological position for the rest of the operation. An arc-shaped incision was made in the perineum, nearer to the anus and the rectum mucosa was separated to about 3 cm. behind the sphincter ani. Under the control of the finger the rectal ventral wall was incised sideways about 2 cm. In this way a connection was established with the lower end of the tunnel where the distal part of the bladder rested. Then the new urethra was pulled out easily by the threads attached to it ; the threads were clamped with forcepts and drawn out. The end of the new urethra was sutured to the skin incision with single catgut stitches which were reinforced at places with single silk sutures. A rubber catheter was placed in the urethra orifice for a few days.

The patient was reanimated all through the operation. The post-operative period proceeded comparatively smoothly. The patient was given moderate doses of antibiotics and cardiac preparations. At the end of the first week the cavity of the new bladder was rinsed out with physiological serum to test its future function. The patient managed to hold the injected liquid for several hours. The emptying of the bladder was preceded by calls for defecation. The capacity of the new bladder was 500 c.c.

The second operation was performed on April 29, 1956 at the end of the fourth week. A relaparotomy was carried out under anaesthesia with ether. New adhesions were discovered around the organs in the small pubis as a result of the operation. The natural bladder was opened. Thin catheters were placed in the ureters until they were disconnected. Circular incisions were made on the bladder membrane around the ostea which were separated, together with the ureters and the adjacent tissue. The latter were connected by means of termino-lateral anastomosis with silk stitches in two stages. A rubber catheter was placed in the urethra to draw off the urine during the first week.

In the post-operative period the patient was subjected to intensified medicine therapy, she was given antibiotics and diuretic preparations. No opiates were administered. The urine was secreted normally, but up to the end of the first week it was mixed with large amounts of mucus. The tests showed albumin, mucus

and bacteria. The reaction was alkaline. Six days after the operation the patient was able to hold her urine for short intervals of one to two hours. For the first time she felt a call to urinate, which she had never experienced before. She urinated at increasingly longer intervals, 5 to 6 time in 24 hours after the end of the second week. She did not have to get up at night. On the 16th day after the operation the urine gave an acid reaction, the albumen disappeared, the sediment contained 7–8 leucocytes. The bacteriological test showed several bacteria paracoli. The urea content of the blood was normal throughout the entire operative period. The condition of the patient was grave till the end of the first week. She complained of sharp pains in the back and in the lower abdomen, general weakness and inertia. Her temperature ranged between 38–39°C for a week. In the second week the patient recuperated very quickly and 19 days after the second operation she was allowed to leave the hospital completely recovered.

Ten months later the patient was subjected to a control examination. Her condition was very good, she had gained 16 kg. She had no complaints, she urinated 5-6 times in 24 hours and did not have to get up at night. The average amount of urine was 200–250 c.c. The capacity of the bladder was 400 c.c. without any residual urine. The blood picture and the sedimentation rate were normal. Several tests of the urine showed : alb. negative, acid reaction, sugar negative, sediment 3–4 leucocytes and fatty degenerated cells, blood urea 20 mgr. per cent. Venous pyelography and ureterography normal. The new bladder had assumed an oval shape. (Figs. 2 & 3).

The second patient, M.I.I., 46 years old, a farmer, has had the complaint from birth, ecstrophy of the urinary bladder. The anomaly affected the pubic bones, the symphysis was not closed, there was no navel, the penis was hypertrophied and had no urethra. (Figs. 4 & 5).

He was admitted to the hospital on April 9, 1957, with a temperature. The clinical examination showed cystopyelitis and bronchopneumonia of the right lung which extended the preoperative period to 20 days.

The first stage of the operation was performed on April 29, 1957. The same operative method was applied as in the first case. In the second stage, performed on May 17, the ureters were

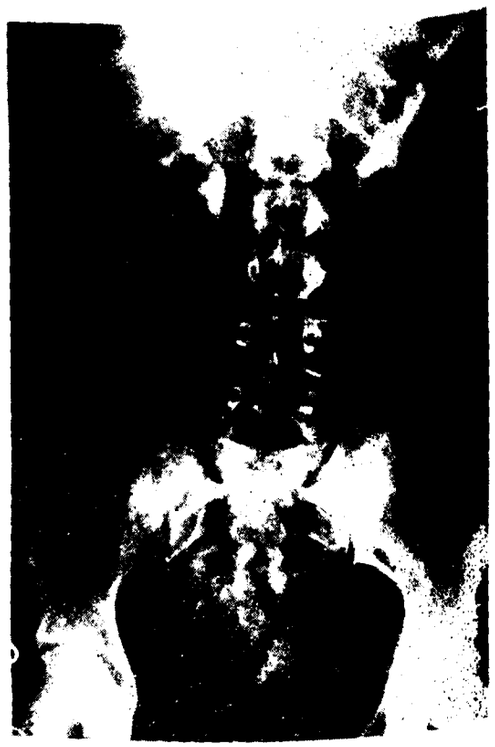

Fig. 2
Venous pyelography of our first patient, after 10 months.

implanted by cutting them above the bladder, because the mucosa of the latter was hypertrophied and unsuitable for anastomosis. The post-operative period proceeded as in the first case. The febrile period also lasted about a week. The administration of antibiotics was stopped in spite of the high temperature, because it was established that the bacteria coli isolated from the urine were resistant to antibiotics and sulphamides. The patient received cylotropin orally with very satisfactory results. The temperature dropped on the day following administration. The urine remained alkaline during the entire period of hospitalization, but the sediment became nearly normal, its rate, however, remained fast. 35 mg. Blood urea was normal throughout hospitalization.

The wound drained in its lower end up to the third week as a small gauze drainage was placed subcutaneously in place of the extirped bladder. After 25 days, the patient was discharged from hospital completely recovered.

Fig. 3
The new bladder had assumed an oval form.

Fig. 4
Ecstrophy of the bladder of the second operated patient.

Fig. 5
Anomaly of the pelvis bones of the same patient.

In both cases the first stage of the operation was performed in 2 hours and the second in an hour and a half.

Our third patient Hristo Ivanov Popov, 11 years old, operated by the same method, completely resembled in his anomalies our second operated patient.

Conclusion

1. The operation on three patients with incontinence of the urine, in which cystectomy was effected and a new bladder made of the large intestine, gave very good results.

2. The following should be watched during the operation :

(*a*) The operation should be performed in two stages to avoid operative shock and to obtain better results ;

(*b*) the tunnel should be made first ; care should be taken not to injure the wall of the rectum, which may result in fistulae.

(*c*) In resection of the intestine, care should be taken not to injure the blood vessles which may lead to necrosis and failure of the whole operation.

(*d*) In transposing the bladder the length of the mesenterium should be sufficient for the perineum to be reached without too much stretching.

(*e*) The separation of the ureters should be done atraumatically, catheters should be placed in them before separation.

(*f*) Anastomosis of the new bladder should be effected terminolaterally to avoid stenosis ; the suturing should always be done with silk thread.

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LATE RESULTS OF HEPATIC AND SPLENIC ARTERY LIGATION IN CIRRHOSIS OF THE LIVER*

BY

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Four years ago, the author presented before the Association of Surgeons of India, his experiences with Portal Hypertension, caused by Cirrhosis of the Liver[1]. Particular stress was then laid on the beneficial results of ligating the hepatic and the splenic arteries. In our later series, the scope of the operation was extended to include the left gastric artery also. This procedure is now known as the "Triple Ligation." As this is a comparatively new method in the field of surgical treatment of cirrhosis, its early as well as late results are worth considering.

Having noticed the dramatic improvement after this operation in our first case of a very advanced cirrhosis with ascites, we decided to try it on similar cases, in a special ward at the K.E.M. Hospital, Bombay.

A great majority of our cases were late ones, with severe decompensation, especially ascites. Each patient was put on the medical regime, as suggested by Patek[2], after a thorough investigation. The progress of the case was assessed at the end of 6 to 8 weeks. If no clinical improvement was noticed, the patient was subjected to the operation of triple ligation, along with the same medical regime. If any improvement was noticed after the operation it was attributed to the surgical intervention.

Altogether 62 cases were admitted to this ward. Fourteen of them improved on the medical regime alone. Eighteen patients went downhill or developed some complication and died. Thirty patients were subjected to this operation. Their early as well as late results form the basis of this report.

In order to appreciate the rationale of this operation, a short review of the salient features of the disease, along with the experimental work will be helpful.

Some unknown toxins destroy the parenchymatous cells of the liver, which are replaced by fibrous tissue. Here and there, some cells escape the ravages of the disease. They regenerate and produce nodules, not only in the substance but on the surface of the organ. This, along with the contraction of the fibrous tissue, gives the liver, its characteristic appearance. Such regeneration of the parenchymatous cells is only possible in the liver.

Normally the branches of the hepatic artery and those of the portal vein, traverse through the liver in the perilobular spaces. They do not communicate with each other, until they pour their blood into the common channel, the sinusoid, in the lobule. From here the mixed blood is taken to the central hepatic vein, which empties into the inferior vena cava. In the wide perilobular space, the high pressure of the hepatic artery is not transmitted to the portal vein with low pressure. As the fibrous tissue contracts, however, and the organ gets smaller, a fair amount of resistance is offered to the vessels passing through. Nature attempts to get over this by producing collaterals, within as well as outside the liver. Some abnormal communications between the artery and the vein are also formed. The portal vein, because of its low pressure, suffers most from the obstructive fibrosis in the organ.

Herrick[3] and Dock[4] carried out perfusion experiments and proved that in a cirrhotic liver, high pressure in the hepatic artery definitely interferes with the free flow of blood through the portal bed, due to lateral transmission of pressure and the formation of arteriovenous fistulae. Dock stated that " in healed alcoholic cirrhosis with ascites and danger of fatal haemorrhage a procedure to reduce hepatic artery inflow may be worth consideration." Berman[5, 6, 7] confirmed this and also proved that ligation of the hepatic and the splenic arteries in portal hypertension not only lowers the pressure, but allows more blood to flow through the diseased organ.

About 30% of the blood in the portal vein comes from the spleen. Ligation of the splenic artery, will not only reduce this load from the portal circulation but will allow more blood from the intestines to reach the liver.

*(Presidential address delivered at the 19th Conference of the Association of Surgeons of India, at Nagpur, on 28-12-57.)

Although 20% of the oxygen supply of the liver is from the hepatic artery and 72% from the portal vein, sudden interference with the arterial supply at the porta-hepatis is probably not compatible with life, even in the presence of anti-biotics. But ligation of the same at a distance, allowing some collaterals in between is quite a safe procedure. We have seen this in many of our cases. In some of the cases who died after this operation, the post-mortem did not reveal any necrosis of the liver.

Two serious complications of the disease are haemetemesis and ascites. Bleeding from the oesophageal varices is the result of portal-hypertension. The rational line of approach is to reduce the pressure in the portal bed by various shunt operations. That form of treatment is not under consideration in this paper.

The causes of ascites in cirrhosis are many. Some of the important ones are :

(1) Sodium retention.

(2) Portal hypertension.

(3) Lymph stasis as a result of Hepatic congestion.

(4) Low Serum albumen.

(5) Presence of inactivated hormones from adrenals, pituitary and sex glands.

None of these, alone, is capable of producing ascites in a cirrhosis patient, but a combination of two or more are at work in a given case and they perpetuate the condition.

Experimental work on the production of massive ascites, and the influence of graded ligations of the hepatic artery on this offer some explanation of the beneficial results we have seen in our cases.

Occlusion or narrowing of the thoracic portion of the inferior vena cava, has produced massive ascites in animals within a period of 2 to 3 weeks.[8] Ligation of the portal vein at the porta hepatis or extra-hepatic thrombosis of the portal vein, fails to produce ascites. Even in cases of intra-hepatic thrombosis of the portal venous bed, no ascites occurs. But when thrombosis spreads to the hepatic veins, ascites follows. Clinically in the rare Budd—Chiari syndrome, there is an obstruction, complete or partial, of the hepatic veins. From these clinical as well as experimental observations, it is postulated that the primary factor for the production of ascites is a block in the outflow tract, the hepatic vein, and not in the inflow tract, the portal venous bed.

In acute ascites, it is the swelling and oedema of the perenchymatous cells of the liver, which compress the hepatic veins. Medical treatment relieves the oedematous condition of the cells and ascites disappears. But in chronic irreversible ascites, the cause is obliterative fibrosis of the hepatic veins. In the liver, the hepatic venous bed is the common outflow tract for the hepatic artery and the portal veins.

Laufman[9] produced ascites by narrowing the thoracic portion of the inferior vena cava, and carried out hepatic artery ligations in stages until all available arterial supply was removed. His results indicate that ascites accummulation cannot be prevented when only the hepatic artery is ligated, but can be prevented in surviving animals when all of the main arterial supply is severed. This suggests that one must produce diminution in the hepatic vein pressure in order to lessen the formation of ascites. We have tied the hepatic artery above the gastro-duodenal branch in all our cases. In successful cases it is presumed that sufficient reduction in the hepatic vein pressure was achieved, to dispel the ascites. While in the unsuccessful ones further distal ligations of the artery are indicated. Michels[10] has shown that there is a great variation in the anatomy of the hepatic artery and its branches in man. Some of the unsuccessful results may be due to this. It is interesting to note that in spite of many causes of ascites the cycle of its formation can be broken by an alteration in only one of them, *viz.*, the vascular factor.

What do we expect to achieve by ligating the three arteries in cirrhosis ? On theoretical as well as on experimental grounds, the ligation is supposed to lessen portal hypertension, allow more blood from the intestines to reach the liver and thus help in regeneration of the cells. This operation may well be classed as a physiological procedure. Are all the criteria fulfilled ? If improvement in the sense of well-being, disappearance of ascites along with other symptoms of cirrhosis and gradual return of liver function tests to normal, are any indications at all, then one must admit that the above criteria are fulfilled. Four of the causes of ascites can be eliminated by arterial ligations.

Post-operative results of Hepatic and Splenic artery ligation in Cirrhosis of Liver

		Early results (2–3 months after operation)			Late results (3–6 years after operation)		
	No.	Status Quo	Good	Died.	Good	Worse	Died
Ascites	20	12	2	6	9	2	2
Ascites Varices.	8	2	1	5	1	...	2
Varices	2	...	2	...	...	...	...

Although it is gratifying to note that the ascites disappeared in 14 cases out of 28, within a few weeks after the operation, we were at a loss to account for failure in others. It was also disquieting to learn that 4 of the good cases from the early follow-up became worse and developed ascites followed by death in two. Cases who did not improve after the operation died within 12 months after the surgical intervention. Once a cirrhotic patient becomes decompensated with formation of ascites, death is certain within a period of a year. Any measure which will check the progress of the disease is worth consideration. The very fact that 10 out of 28 severely decompensated cases enjoy normal health 3 to 6 years after this operation, is enough to convince a sceptic about the value of this operation.

Realisation of this alone prompts one to recommend this operation to you. Unfavourable reports in the literature are based on the experience of one or two cases only[12]. It should be realised that this operation is still in an experimental stage. With more experience about the selection of cases, I am confident that we would be able to improve the figures. Sheila Sherlock[13] in her latest book on "Diseases of the Liver and Biliary System" states that 'Hepatic arterial ligation has nothing to commend it.' The author is afraid that he does not share this view at all. Experimental evidence may not be entirely convincing and we may not be able to account for our failures adequately, but the glaring fact is the clinical improvement noticed not in a few but in many. One could go still further and state that even if one gets a good result in an occasional case, that life is definitely saved and the operation has justified itself. At the cost of repetition, it may be mentioned that we carried out this procedure only after a thorough trial of conservative medical treatment.

It is not an easy operation by any means, but not a very difficult one either. It can be carried out by any surgeon at a district hospital. Four things are very essential: (1) Blood transfusions at least two, (2) good light, (3) Suction apparatus and most important of all (4) patience. If a reliable anaesthetist is not available, the operation can be carried out under intercostal block. It takes nearly 2–3 hours to perform this operation.

While not intending to give the details of the technique of the operation one would like to stress some important points in view of our experience. An access through the gastro-colic ligament to the lesser sac will give a direct approach to the pancreas. An incision through the peritoneum of the posterior abdominal wall, along the entire superior border of this gland, is a very helpful step. This will mobilize the gland and enable the assistant to retract it downwards during the search for the arteries. In a cirrhotic patient the sight of tremendous formation of collaterals in this space is frightening. The situations of the arteries are recognized by palpating for their pulsations. Some collaterals have to be sacrificed before the arteries are exposed. Sudden disappearance of pulsations in these blood vessels is not unusual. This is probably due to arterial spasm, as a result of handling. Fortunately it lasts only for a minute or two. Identification of the artery is facilitated by the presence of the network of sympathetic fibres in the wall. Once one of the arteries is found out, it should be traced to its origin when identification of the other becomes easy. A wedge from the edge of the liver is removed for biopsy in each case before the abdomen is closed.

Although we had instructed the patients to maintain the same diet, with high proteins, after going home, we were surprised to note that very few of them observe the rule. Sodium restrictions were not tolerated by any, and none of them refused to take any food without salt. All of them did avoid very spicy foods, fried things and fats. Later these patients reverted to their original diet without developing any trouble.

After watching these patients over a prolonged period some suggestions about the selection of the cases suitable for the operation can be offered. Those with ascites alone as the only complication, without oesophageal varices on screening, respond well, immediately as well as in the long follow-up. Cases with both ascites and varices had the highest mortality. Only one patient out of 8 is alive and well three years after the operation. Although the portal veins seem to have narrowed, as shown by pre and post operative splenoportograms there is no suggestion that there is any change in the varices. We have had only two cases of varices alone in this series. Both of them had attacks of haemetemesis before operation. One of them still bleeds after the surgery and no kind of interference is allowed by the parents. The other one is remarkable. This middle aged man had seven bouts of haemorrhage during a period of three years before the operation. Triple ligation was done, early in 1954. It is now $3\frac{1}{2}$ years since the operation and one is glad to mention that there has been no attack of bleeding so far, and the patient is hale and hearty. The oesophageal varices however are still there and of the same magnitude as judged from X-Ray films.

Pre-operative administration of antibiotics is not necessary in man. In some of our cases this was not given due to oversight but apparently no harm has followed.

This operation is not recommended in an acute emergency. Apart from the general low condition of these patients, there is one contra-indication, in our experience, and that is the presence of jaundice noticed clinically or suspected by high icteric index.

The disease is quite common in this country and the mortality is 100% when decompensation sets in. As long as the etiology remains obscure, our primary object is to bring abou amelioration in the symptoms. Our result have shown that not only is this possible but the general condition of the patients improves so much that they are apparently cured.

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ADAMANTINOMA JAW

BY

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Adamantinoma is a tumour peculiar to the jaws because of the element of odontogenesis in it, though it has been found to occur in the Tibia and the Pituitary gland. It is not a rare tumour but the diagnosis is difficult and controversial and has to be made by an expert Histopathologist.

Morbid Anatomy

Adamantinoma is usually a central tumour but cases are on record which are of peripheral origin. In the mandible it appears as a hard mass expanding the jaw. As it gains size, it may ulcerate into the mouth and gets secondarily infected.

Section of a typical specimen shows large and small cystic areas filled with a brownish fluid or gelatinous material. Other areas consist of more solid tissue-soft or firm in consistency and usually whitish in colour. The growth may present as a fleshy or cauliflower projection into the mouth or as a solid mass of tumour without cyst formation. The tumour may weigh as much as 1500 Gm.

Histology

A typical specimen shows Masses of epithelial cells, peripheral ones being columnar and arranged in palisade fashion and the central stellate cells in a reticular fashion corresponding to the reticular cells of a normal enamel organ. Stroma consists of very loose areolar tissue or is more often cellular. Blood vessels are frequent. Central areas of epithelial clumps often become cystic and all stages of cyst formation may be seen. In other cases the epithelial cell arrangement resembles that of basal-celled-carcinoma of the skin, like Rodent ulcer. Frequently epidermoid features are present *e.g.* epithelial pearls or prickle cells. Mitoses may be seen in the more malignant type and changes due to infection and hemorrhages may be present.

Campbell[1] observed in two cases and Sirsat[2] in one case, presence of large granular cells, brightly eosinophilic and occurring singly as a syncitium or as sharply demarcated areas of various sizes amongst typical adamantinomatous tissue. Transition between granular stellate cells and between the granular and columnar cells were also seen. These findings give the appearance of a Myoblastoma in the adamantinomatous tissue. This observation may have no more significance than that of coincidental occurrence of two unrelated lesions or they may be also interpreted as a peculiar variant of an uncommon type of adamantinoma, hitherto unnoticed.

Histo-pathological variations of adamantinoma

Depending upon the different varieties of histology encountered in the adamantinomas the following variations are seen :—

1. *Epithelioma type :* The cells are poorly differentiated and have great infiltrating power. It is usually a solid type of growth.
2. *Stellate type :* It is made up of stellate cells as seen in the developed enamel organ.
3. *Ameloblastic type :* It is made up of follicles in which the peripheral cells reach the cylindrical or ameloblastic shape. The so-called basal.—celled carcinoma of the jaws is closely related to this type. It rapidly grows, tends to metastasize and also recurs early and easily.
4. *Acanthoma type :* It shows epithelial pearls and prickle cells in the reticulum.
5. *Adamantino-carcinoma :* In this type a carcinomatous area is seen developing in the adamantinoma.
6. *Melanotic type :* This variety shows epithelial cells and the space between them has pigment which gives the tumour a bluish black discolouration in the centre.
7. *Haemangioadmantinoma :* It shows the stroma and epithelial tissue surrounding blood filled spaces.
8. *Adenomatous type :* In this variety the epithelium tends to differentiate into glandular structure.

Pathogenesis

Lewis[2] and Zegarelli[9] discussed the theories of origin of adamantinoma which are as follows :—

1. *Residual epithelial cell rests :* These have been described by Malassez.[3] Proliferation of these cells is thought to give rise to adamantinoma.
2. *Anomalies in the process of proliferation of the enamel organ :* Anomalies in the proliferation of enamel organ are supposed to give rise to the development of Adamantinoma. Enamel organ has various layers, but it is uncertain from which layer the tumour originates.
3. *Mucous membrane epithelium of the jaws :* The tumour is supposed to originate in the basal layer of the epithelium covering the alveolar process.
4. *Epithelium of the odontogenic cysts or the epithelial remnants in the periodontal membrane :* It is unlikely that all the tumours originate thus though adamantinematous tissue might develop in the cyst wall.

Thoma[7] showed examples of all the four possibilities. Zegarelli[9] convincingly showed the origin of the tumour from the group of cells forming the outer layer of the enamel organ but Schulenberg[4] feels that these cells correspond to the basal or germinative layer of cells, from which the enamel organ originates. Thus he feels that it may be called a basal-celled tumour.

If the origin of adamantinoma from the basal cells is accepted then the pathogenesis of extra oral adamentinomas also becomes clear.

So, according to Schulenberg adamantinomas are examples of basal-celled carcinoma originating from the epithelial cells of the embryonic cell-rests or those driven into the periosteum by trauma. Some types of this tumour are not distinguishable from the so-called basal-celled carcinoma of the maxillary antrum and this fact supports Schulenberg's hypothesis.

The following clinical features in a series of seventeen cases are being described :

Age incidence : The youngest patient was only 15 months old. Considering the duration of 10 months in this case, it may be reckoned that the disease started in the patient only at 5 months of age. Very few cases are recorded at such a young age.

The eldest patient was 36 years and the majority of cases (11 of them) were below 20 years.

Sex incidence : Out of the 17 cases, six were females.

Social status : Almost all cases belonged to the middle or lower class.

Religion : Only one out of the seventeen cases was Muslim, the rest being Hindus.

Duration : It ranged from 2½ months to 6 years.

Site : Maxilla was involved in 3 cases and mandible in 14 cases, the site and distribution being as follows :

		Cases
Mandible :	Right	7
	Left	3
	Symphysis-menti	2
	Whole mandible	2
Maxilla :	Right	2
	Left	1

Teeth were found missing in cases of dentigerous cysts diagnosed by radiography. In other cases the teeth were displaced, loose or missing due to the invasion by the growth.

Ulceration and secondary infection, pus discharge and pain was found in 9 out of the 17 cases as these sought treatment appreciably late.

X-ray findings

Radiographic diagnosis of adamantinoma was definite only in six cases.

Unerupted tooth into the cystic cavity was reported in the skiagram of 3 cases only and in one case the roots of teeth were reported to be projecting into the cavity. Histo-pathologically all these cases were adamantinomas.

Cases were misdiagnosed by radiography prior to operation as follows : --

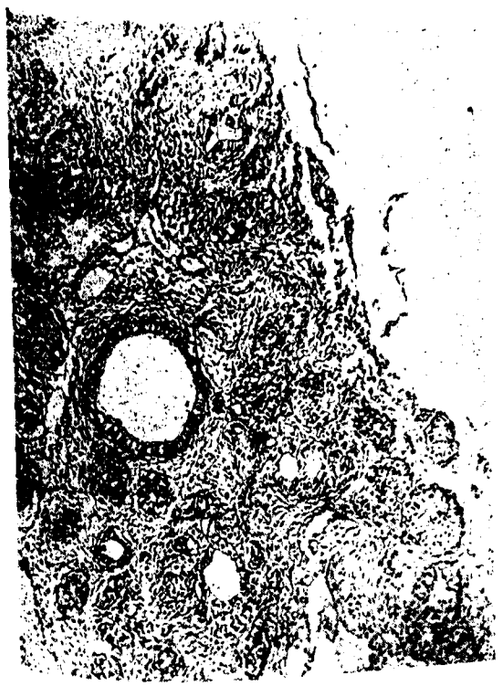

Fig. 1
Case No. 1. Satish Chand.
Microphotograph showing solid strand of cells with formation of cysts in adamantinomatous tissue.

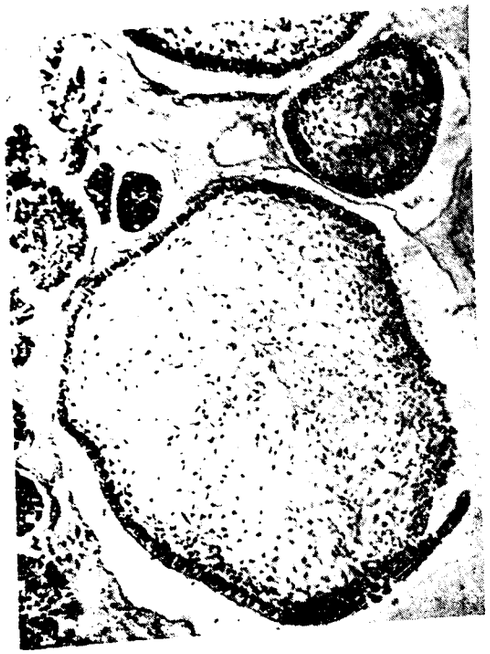

Fig. 2
Microphotograph of Case No. 7 showing the Stellate reticulum surrounded by layer of columnar cells.

Fig. 3
Chandra Pal. Case No. 2. Skiagram appearance P.A. view. Note the soft tissue swelling shadow and the tooth lyin in the maxillary antrum (right).

Fig. 4
Lateral skiagram view of the same patient.

Fig. 5
Krishna Gopal. Case No. 10.

Fig. 7
Skiagram of the same patient 7 months after operatio of curetting. Note the cysts in the jaw.

Fig. 6
Skiagram-Lateral View—of the mandible. Note the multiple cysts and an unerupted tooth (Case No. 10).

Fig. 8
Kishan Bir Singh. Case No. 11 (Adamantinoma).

	Cases
Osteoclastoma	2
Dentigerous cyst ...	4
Dental cyst	2
Simple cyst	1
Radio-opacity of maxillary sinus	1
Inconclusive	1

Biopsy

The final diagnosis depends on a suitable biopsy. In the present series radiographic and biopsy diagnosis coincided only in six cases.

In case No. 3 the skiagram suggested osteoclastoma, the biopsy from the growth by aspiration needle twice proved epidermoid carcinoma grade III. Finally, the biopsy of the material removed at operation was adamantinoma with epidermoid carcinoma like structure. Such a case has also been reported by Willis.[8]

In case No. 10, clinically dentigerous cyst of mandible, the skiagram also suggested the same diagnosis but the biopsy report was adamantinoma. The second biopsy after a fortnight revealed epidermoid carcinoma grade II. The third biopsy of the resected mandible showed adamantinoma with changes of Anaplastic Carcinoma (Epidermoid).

In case No. 11, clinically adamantinoma mandible, three biopsy reports were fibrous epulis, but the biopsy of the material removed at operation proved to be adamantinoma.

The above cases stress the need and importance of repeated and deep biopsies in doubtful cases.

Biopsy wound healed in all cases as was also observed by Schulenberg.[4]

Fig. 9
Same case after operation of removal of 3/4th of mandible.

Fig. 10
The specimen of jaw removed from the above patient.

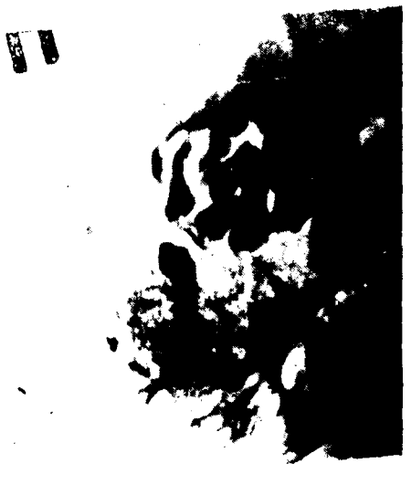

Fig. 11
Skiagram appearance of Case No. 11. Adamantinoma (Kishan Bir Singh).

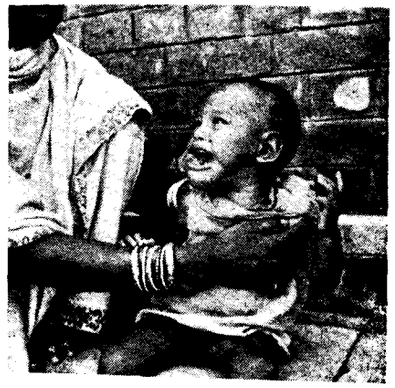

Fig. 12
Prem Nath. 15 months old patient, the youngest patient of the 17 cases. (Case No. 12).

The following precautions should be observed while taking a biopsy :—

1. Biopsy of oral tumours including adamantinoma should be sufficiently deep as a superficial biopsy may lead to an erroneous diagnosis of basal-cell carcinoma or cylindroma.
2. Biopsy should always be repeated in doubtful cases. The importance of repeated biopsy examinations is very well illustrated in cases 3, 10 and 11 of the present series.
3. Histopathological differential diagnosis may present difficulty in some cases, especially in the mucus and salivary gland tumours as well as mucus secreting and cystic epidermoid carcinomas and so adamantinoma should always be differentiated from these. A thorough histopathological examination of more than one section from the tumour might clinch the diagnosis in doubtful cases.

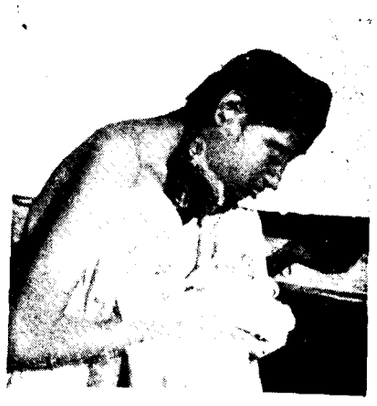

Fig. 13
Rajeswari Devi. Case No. 13. Ulcerating adamantinoma.

Fig. 14
Skiagram appearance of the lesion in the lower jaw of the above patient. Note the increased thickness of bone and its expansion

Fig. 15
Ramvati. Case No. 14. Note the swelling of the right lower jaw.

Fig. 16
Skiagram appearanee in 1954. Note the huge cystic expansion of the mandible.

Fig. 17
Skiagram appearance about 2 years after (Nov. 1956)

Fig. 18
Vimla. Case No. 17. Adamantinoma of left mandible.

Fig. 19
Skiagram appearance of the mandibular swelling. Note the great cystic expansion and trabeculations in it.

Carcinomatous change was seen in cases 3 and 10 only. Metastasis was found in the lymph nodes in cases Nos. 11 and 16.

Treatment

The standard treatment is complete resection of the part with the growth, as the tumour is radio-resistant.

Three cases gave history of previous operations (the exact nature of operation being unknown) before seeking treatment in this hospital.

Partial resection of mandible was done in case No. 3 who had involvement of the whole mandible with gross enlargement of the chin. The bone in the region of chin was removed for cosmetic reasons after deep X-ray therapy had reduced the size of the growth.

Hemi-mandibulectomy was done in five cases Nos. 5, 7, 10, 14 and 15. In case No. 11,

Observations on Adamantinoma by Different Workers

No.	Author	Total cases	Age	Sex	Duration	Site	Malignancy	Recurrence
1	Robinson, 1937.	379	Average 30 years (at time of discovery).	Male 142 Female 169	Average 8.5 years	... 293 cases ... ... Mandi. 84% Maxil. 16%	4.5%	119 cases.
2	Geschickter and Copeland, 1949.	43	10—35 years (70% of cases).	No mention ...	5—50 years ...	Mandi. 36 Maxil. 7	1 case with metastases.	Majority, one or more times.
3	Schulenberg, 1949.	88	Around 40 years.	78 cases— Male 42 Female 36	No mention	... 67 cases— ... Mandi. 79% Maxil. 21%	All malignant only a question of degree.	No mention.
4	Anders Sonesson, 1950.	39	13—72 years ...	Male 13 Female 26	No mention	... Mandi. 34 ... Maxil. 5	None of Carcinoma One of Sarcoma.	14 cases.
5	Sirsat, 1952 ...	15	9—60 years ...	Male 11 Female 4	Up to 18 years... 6 of less than one year.	Mandi. 14 Maxil. 1	No mention ...	No mention.
6	Srivastava, S. P. and Sikroria. 1957.	17	1¼—36 years	Male 11 Female 6	¼—6 years ...	Mandi. 14 Maxil. 3	2 cases. Two with metastasis in lymphnodes.	Two cases with carcinomatous change in adamantinoma who were treated by curetting only.

NOTE : Some percentages have not been made out because of inadequate number of the cases.

Personal Series of Adamantinoma Cases

No.	Name	Age and Sex	Admitted	Duration	Site	Teeth	Ulceration and secondary infection	Skiagram	Biopsy	Treatment	Type	Metastasis and mortality
1	S.C.	11 HM	25-11-1950	3 months.	Mandi. Rt.	Normal.	Nil	Dentigerous Cyst.	Adamantinoma with solid strand of cells and cyst formation.	Curetting	Cystic	...
2	C.P.	16 HM	15-12-1950	7 months.	Maxil. Rt.	Upper Rt. Canine missing.	Nil	Dentigerous Cyst.	Do.	Do.	Do.	
3	G.	20 HM	12- 3-1951	6 years	Whole mandible.	Edentulous.	Nil	Osteoclastoma.	On aspiration of growth twice—Epicarci—operated specimen : Epicarcinoma Finally admanti. with Epi-carci like structure or *vice versa*.	Deep X-ray and partial removal of mandible and symphysis menti.	Solid	...
4	L.S.	25 HM	12 -3-1951	2½ months.	Symphysis menti.	Normal	Nil	Cyst in the region of symphysis menti	Admanti : with solid strand of cells and cyst formation.	Curetting	Mono Cystic.	...
5	M.	18 HF	11- 2-1952	4 months.	Mandi. Rt.	Normal	Nil	Dental Cyst.	Do.	Resection	Do.	...
6	M.S.	34 MM	1-12-1953	10 months.	Maxila and Palate Rt.	1 and 2 Rt. upper molars missing.	Present.	Dentigerous Cyst.	Adeno-Admanto-blastoma.	Too advanced.	Solid	...
7	U.	16 HF	29- 1-1954	2 years	Mandi. Lt.	Lower Lt. and premolar missing.	Present.	Admantinoma.	Do.	Resection	Solid as well as Cystic.	...
8	R.P.	30 HM	29- 7-1955	1 year	Mandi. Lt.	Extraction of carious tooth.	Present.	Simple Cyst.	Andmantinoma	Curetting	Mono Cystic.	...
9	B.S.G.	28 HM	12-12-1955	2 years.	Symphysis menti.	Normal	Nil	Osteoclastoma.	Do.	Do.	Polycystic.	...

Personal Series of Adamantinoma Cases—(*Contd.*)

No.	Name	Age and Sex	Admitted	Duration	Site	Teeth	Ulceration and secondary infection	Skiagram	Biopsy	Treatment	Type	Metastasis and mortality
10	K.G.	9 HM	7- 4-1956	3½ years.	Mandi. Rt.	Normal	Nil	Dentigerous Cyst.	Admantinoma from cyst wax with carcinomatous changes. Biopsy of resected specimen :— Adamanti with Anaplastic Carci (Epidermoid).	Curetting in 1st operation and Rt. Hemi mandibulectomy after one year.	Mono Cystic.	Recurrence after curetting.
11	K.B.S.	HHM	21- 7-1956	6 months.	Mandi. Rt. and symphysis.	Displaced and loose.	Present.	Admantinoma.	Fibros epulis in biopsy. Operated specimen : Adamantinoma.	Deep X-ray followed by resection of ⅓th of mandible.	Solid	Lymph nodes were enlarged in submaxillary region.
12	P.N.	15 mths.	26- 7-1956	10 months.	Mandi. Rt.	Do.	Present.	Inconclu. ...	Adamantinoma	Palliative deep rays.	...	...
13	R.	16 F	19- 9-1956	11 months.	Do.	Molar tooth extracted loose teeth.	Do.	Adamantinoma.	Do.	Too advanced for resection.	...	...
14	R.W.	20 HF	11-11-1956	2 years	Do.	Rt. last lower molar missing tooth loose and displaced.	Do.	Do.	Do.	Resection	Solid as well as cystic.	...
15	R.D.	22 HF	25- 4-1957	3 years	Do.	Caries of Rt. last lower molar tooth extraction.	Nil	Do.	Do.	Do.	Solid	...
16	B.S.	36 HM	9- 7-1957	1 year	Maxil. Lt.	Extraction of left upper canine Adjacent incisor missing.	Present.	Radio-opacity of Maxillary Sinus.	Do.	Resection c block dissection of supra hyoid lymph nodes.	Do.	Metastasis in cervical lymph nodes.
17	V.D.	17 HF	O.P.D.	1½ years	Mandi. Lt.	Nil.	Present.	Adamantinoma.	Do.	Deep x-rays (refused operation).	Do.	Nil.

3/4th of the mandible was removed after deep X-ray therapy had been applied to the growth.

Maxilla was removed along with supra-hyoid dissection of lymphnodes in case No. 16.

Curetting was done in six cases Nos. 1, 2, 3, 8, 9 and 10 which were all cystic. The biopsy of the curetted material revealed adamantinoma, except in cases Nos. 3 and 10 where epidermoid carcinoma was detected. Case No. 10 was aged 10 years and had anaplastic carcinomatous changes also.

Four cases Nos. 6, 12, 13 and 17 were too advanced for surgery and so were advised palliative deep X-ray therapy.

Discussions and conclusions

The table given above gives an idea about adamantinoma as studied by various workers with wide variations in the observations. Nevertheless, adamantinoma is a disease which affects the younger age group, the lower jaw and the middle or lower class of people according to our observations.

Though the study does not conclusively prove any definite theory of origin of adamantinoma yet it is felt that the abnormality in the development of enamel organ has an appreciable role in the pathogenesis of adamantinoma as its relationship to the dentigerous cyst also suggests it. Site of 3rd molar also suggests relationship to an aberrant tooth germ at the angle of the jaw. These tumours may also be taken to be examples of primary basal-cell carcinoma originating from epithelial cells of the embryonic cell-rests or those driven into the periosteum by trauma.

The presence of large granular cells in adamantinoma as observed by Campbell and Sirsat was not seen in the present series.

Pain appears to be only a secondary symptom in adamantinoma and the majority of the patients seek medical aid only because of an increasing swelling. In neglected cases and those coming late the growth ulcerates with the occurrence of bleeding.

Adamantinoma should be considered a malignant tumour and dealt by resection of the growth. In the present series this could not be done in all cases due to the patients not agreeing and so only curetting was done or Deep X-ray was advised. Some cases were too advanced for resection.

Radiotherapy has no curative value in adamantinoma. It may reduce the size of the growth when malignancy has occurred, otherwise its effect on this tumour is of doubtful value.

Recurrence in most cases of adamantinoma is due to incomplete removal. It is felt that recurrence should be judged after observation for a minimum period of 10 years.

The distinction of solid and cystic types of adamantinoma serves no purpose and there is no conclusive evidence that the solid type is more malignant than the cystic. It is only of pathological importance.

The name of adamantinoma is controversial, but it is felt that it may be called adamantoblastoma with the histological picture of basal-celled carcinoma.

All the odontogenic cysts are potentially malignant and it is suggested that their lining wall should be subjected to thorough histopathological examination for adamantinoma.

Summary

A personal series of 17 cases of adamantinoma is described along with its morbid anatomy and pathogenesis.

The list of the cases studied is given along with the observations of other workers.

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We are grateful to Dr. Parmanand, Superintendent, Sarojini Naidu Hospital, Agra for allowing us to publish the hospital records. We are very thankful to Dr. P. N. Wahi, Professor of Pathology, Medical College, Agra for the diagnosis of the specimens and to Dr. P. K. Haldar, Head of the Department of Radiology, Medical College, Agra for the interpretation of the skiagrams.

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HERMAPHRODITISM

BY

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The phenomenon of a combination of both the male and female sex characteristics in the same individual has been termed hermaphroditism. Klebs classified this into true and pseudohermaphroditism which has been accepted by many authorities. A pseudohermaphrodite is one who possesses the glands of one sex but reveals the secondary characteristics of the opposite sex. To understand the intriguing genesis of hermaphroditism, it may be useful to recapitulate the normal process of development of the gonads.

Gonadal primordia appear in the 5 mm. embryo as the thickening of the coelomic epithelium on the medial side of the mesonephros. This is the genital ridge. Consequent to the disappearance of the basement membrane, coelomic epithelium sinks into the mesenchyme producing the genital blastema. Urogenital mesentry, a fold of peritoneum, suspends this from the posterior abdominal wall along with the mesonephros. Further grooves appear separating the genital blastema from the mesonephros and the primordia of the developing adrenals, thus forming mesorchium or mesovarium.

Upto 17 mm. stage, in this process of development, there is no indication as regards the future sex, gonads are unidentifiable as testis or ovaries though the sex of the embryo is determined at the time of fertilisation of the ovum, the sex of the zygote depending upon whether or not the 'X' chromosome was present in the fertilising sperm.

Genital blastema at 13 mm. stage becomes divided into a large number of primary sex cords by fibrous tissue bundles. The secondary germinal cords subsequently appear under the germinal epithelium forming a net work, the rete, which then goes to meet the tubules of the Wolffian body. Various changes of the primary and the secondary cords contribute to the sex of the gonad. Predominance of the primary cords makes the testes ; on the other hand, if secondary cords predominate we get ovaries. In normal development there is suppression of one element by the other. How the abnormal development is brought about is still a matter of conjecture, many theories have been advanced to explain the abnormality.

Theories of Intersexuality

Goldschmidst in his " Turning point " theory says that the primitive cells are bisexual and the sex is determined by the preponderance of one type of sex determining factor in a fertilised ovum over the other, after the male set of chromosomes have reacted with the female sets. In a normal female, the femininity is in overwhelmingly large proportions to the masculinity in embryonal life and it suppresses any tendency to masculinity with vigour. Thus it retains its female characteristics. So it is with its male counterpart, where any inclination towards femininity is resisted. If, however, the difference between the two sex types is not significant or the time may arrive when a sex type which hitherto has been resisted asserts itself after the initial sex type (which has so far accounted for the sex) has exhausted itself in the tug of war. This then is the " turning point " when a genitically female individual, be it in the embryonal stage or in the postnatal life obtains male characteristics or vice versa. According to this theory, the perfection with which the sex reversal occurs is predicted upon the time when this occurs. Those portions of gonads which have already formed, will persist and only those that are not definitely formed, undergo a new transformation in the direction of new sex.

Other geniticists like Bridges and Boltzer, however, reject Goldschmidst's Turning point theory. They suggest that there is no pure male developmental period followed by pure female developmental period in intersexuality. According to Bridge's balance theory a joint action of all the genes of the entire complement of chromosomes produce the feature of an individual. Some of them tend to direct the forces in the opposite direction so as to retard the tempo of development in one particular direction, the resultant form being the fruit of the balance of the opposing forces.

Masculine Hermaphroditism : In this common form of hermaphroditism testes are present in people who are manifestly females and who indeed feel themselves to be so. The testes show the character of cryptorchid gonads with no evidence of spermatogenesis as is only to be expected. Leydig cells are well formed. testes occupy the site of the ovaries or may be found in the hernial sac. Broad ligament may prevent the descent of the testicle or else fused mullerian ducts may do so. Epididymis and vas deferens are often found and a prostate is always present.

Feminine Hermaphroditism : This condition is comparatively rare. The external genitalia may assume very different forms starting from a well developed penis to a hypertrophy of the clitoris.

Case Report

A married woman aged 21 admitted under one of us (Prof. U. P. Gupta) with a history of primary ammenorrhea. Every month she used to get pain all over the body especially in the lower abdomen and small of the back, which used to last for four days. During this period she had white discharge per vaginum and occasional vomiting.

Clinical examination revealed a woman of moderate build and fair nutrition with well developed secondary female sex characters. Breasts and vagina were well formed as can been seen in the photograph. (Fig. 1)

Pubic hair and axillary hair was conspicuously absent. Patient had bilateral inguinal hernia, bigger on the left side, both were easily reducible. Careful examination revealed two solid masses one on each side resembling testicles, which were felt among the content of the sac. Vaginal examination did not reveal the portio vaginalis of the cervix, uterus and adnexa could not be felt. It was decided to do herniorrhaphy on both sides and explore the abdomen to establish the presence or otherwise of the uterus and ovaries.

17th January 1957 under general anaesthesia, oblique incision taken, inguinal canals opened up, first on the right side then on the left, sacs isolated, invaginating the sacs and adherent to it were the (?) testicles with pampiniform plexuses and vasa diferentia.

Sacs isolated and ligated high up, high orchidectomy done and the internal rings closed, herniorrhaphy done and the wounds closed in layers on both sides.

Median subumbilical laparotomy revealed the absence of uterus and adnexa. Vaginal vault was pushed up with a finger per vaginum confirmed the absence of even a rudimentary uterus. Abdomen closed in layers.

Histological examination of the tissue (? testis) by Prof. N. C. Dey, Professor of Pathology—Reference 65/57, dated 31st January 1957.

Histological examination of the two main masses :

These show the structures of testis as evidenced by seminiferous tubules together with interstitial cells and connective tissue stroma. Seminiferous tubules are comparatively smaller with a few sustentacular and spermatogenic cells. Active spermatogenesis is absent. Interstitial cells are few and scanty. There is marked increase of interstitial connective tissue stroma. No ovarian tissue seen.

(2) Section from the small cyst attached to one of the main masses shows fibrous wall with epithelial lining of cuboidal cells.

Fig. 1

Fig 2.

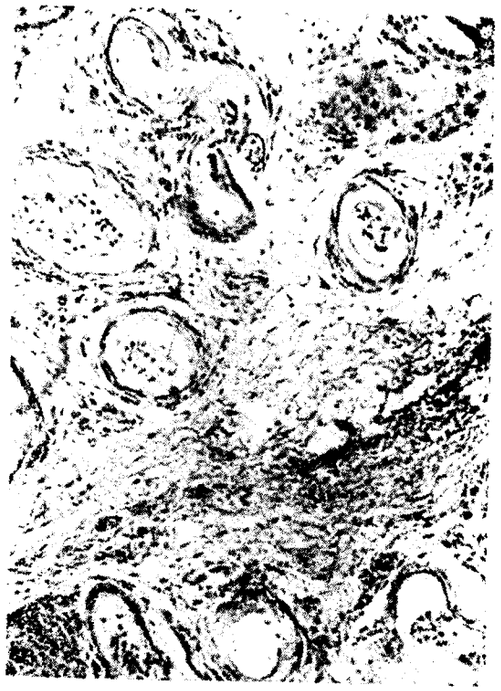

Fig. 3
Section seen under lower powers

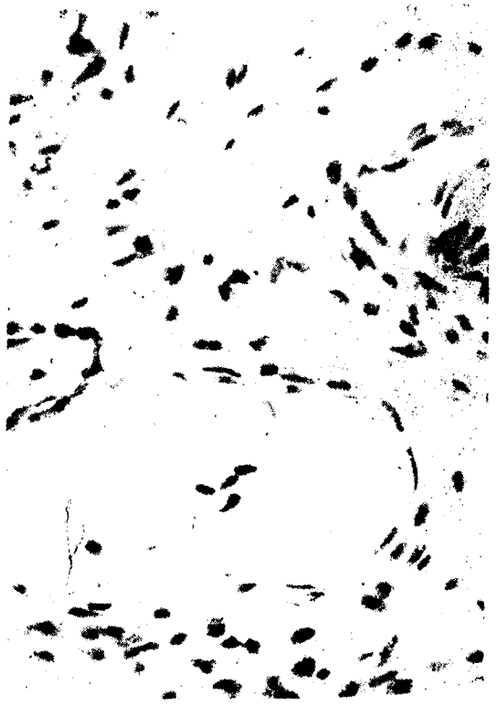

Fig 4
Section seen under high power

It is indeed remarkable that in the absence of any ovarian tissue the secondary sex characters were so well developed.

Our thanks are due to Dr. Pradipta Kumar Das Gupta for the valuable help rendered in getting this paper ready. We wish to thank Prof. B. N. Banerjee F.R.C.S., Superintendent, Assam Medical College Hospital for allowing us to report the case and to Prof. N. C. Dey, Professor of Pathology for the help rendered.

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THE DEFORMED NOSE IN HARE LIP AND ITS METHOD OF CORRECTION

BY

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The appearance of the nose is important in patients with cleft lips and should require close attention in both primary and secondary repairs—the repair of the nose is as intricate if not more so than of the hare-lip. It is well put by Sir Harold Gillis and Dr. Ralph Millard in their latest book on Plastic Surgery—"a well mended harelip would pass unnoticed at a cocktail party were it not for the nose. It is natural for a surgeon to be so pleased with a satisfactory lip result that his eyes go temporarily out of focus upon the nose." To correct the hare-lip nose defect requires a prolonged and detailed experience of rhinoplastic surgery. It is true that no two cases of hare-lip are alike, and more so, no two noses of Hare-lip cases are alike hence the method of repair will differ in each case. Every surgeon has his own views of correcting the lower nose which is the result of long experience and study of results.

It is interesting to notice and study the actual anatomical defect of the nose in unilateral and bilateral cleft lip cases.

1. The floor of the nose is absent in complete hare-lip, and in an incompleted hare-lip it is widened. The maxilla of the affected side is underdeveloped and hence the naso-labial structures on that side are situated slightly posteriorly as compared with the normal side. In cases of double complete hare-lip, the central portion of the lip is attached to the columella of the nose and the premaxilla is rotated forwards. The whole segment is mobile and can be pushed backwards. The columella is very short and underdeveloped hence the tip of the nose is depressed after repair.

2. The lower alar cartilage is flattened and the point of junction of its medial and lateral crura is at a lower level than on the normal side. The angle formed by the medial and lateral crura is wider than on the normal side. The inner surface of the alar cartilage instead of being in the form of a tube and pointing towards the columella is flat and pointing towards the maxilla. The mucous membrane is shortened.

3. The columella is not in the midline but obliquely placed with the tip of the nose deviated to one side. This is due to an obliquity of the cartilaginous septum as a whole and more marked in the region of the columella. The columello-lip angle as seen from the side is generally less than 85 degrees instead of the usual 90 to 100 degrees. This gives a beaked appearance to the nose. In bilateral hare-lip cases the columella is so short that it gives an appearance of a nose as if pressed against a window pane.

4. The maxilla is underdeveloped. Associated with this underdevelopment there are variations in the anatomical form and number of teeth. The appearance of the face is a typically "dished-in" upper lip and apparently pronounced chin. The growth of the lower jaw is usually normal but because of the underdevelopment of the maxilla, an illusion of mandibular prognathism is created. In many cases the width of the upper dental arch is reduced and there is a cross bite of the premolars and molars—the lower teeth occluding outside the upper teeth—quite the reverse of what is found in normal individuals.

When a hare-lip is dealt with first by a competent surgeon the result has a better chance and a standard method will usually suffice. When many others have had a go, the problem then falls on fundamental principles rather than a particular routine technique. A good hare-lip and nose repair requires the following :

1. Nostrils on both sides should appear symmetrical.
2. Slight pouting of the upper lip.
3. Floor of the nose of equal breadth and on the same level on both sides.
4. Absence of oronasal fistula.
5. Absence of notching of the lip margin.
6. Cupid's bow present.
7. No transverse stretching of the upper lip.
8. Faint scar line and absence of irregular stitch marks.

Fig. 1

Fig. 2

Figs. 1 & 2: Unilateral Harelip—showing anatomical defects of the lower nose and maxilla. Note the flaring of the nostril, unequal dome of the nose, and underdeveloped maxilla.

Fig. 3 Before.

Fig. 4 After.

Figs. 3 & 4: Unilateral incomplete harelip with slight spread of nostril floor. Nine months after repair. Defect passed unnoticed at first birthday party!

Fig. 5 Before.

Fig. 6 After.

Fig. 5 & 6: Unilateral Harelip before and after repair. Slight pouting of upper lip and cupid's bow formation. Both nostrils

Defects of the nose noticed in a badly operated hare-lip :—

1. Wide nasal floor. This is more common in operations of complete hare-lip when there is absence of bony support and the soft tissues stretch after primary operation. Very often wide scar tissue is present indicating that though originally correctly repaired, the tissues, *i.e.*, the skin and muscles have stretched. Inadequate undermining and poor approximation of muscle layer may lead to early flaring of the nostril floor.

2. The columella may remain oblique when the septum and the nasal spine have not been corrected at the primary operation.

3. The tip of the nose on the affected side may be at a lower level than the one on the normal side. This is because the medial crus of the lower alar cartilage is shorter than its corresponding one on the opposite side.

4. The nostril also has a droop at the junction of the medial and lateral crura.

5. Depression in the lateral crus of the lower alar cartilage. This depression also produces a bulging or a web in the inner lining of the nostril. Obstruction to breathing is also present is this bulging if marked.

6. Contracture of the nasal lining. After primary repair if there is a loss of lining, then healing will leave behind a web or an actual stricture about one centimeter away from the nostril margin near the alar base. This may follow an intercartilaginous incision with loss of lining mucosa.

Treatment : Although it is impossible to separate the correction of the hare-lip from that of the nose, in this article, the details of the hare-lip part of the operation are purposely omitted. Suffice it to say that either a quadrangular flap repair of Hagedorn Le Mesurier type, has given consistent good results to the majority of plastic surgeons and which the author does as a routine, or any other modification which the surgeon is accustomed with. Any method of lip repair aims at the criteria laid down in the early part of the article. If the result can satisfy those conditions there is no need for a surgeon to go on changing his technique of lip repair. Wider gaps with tissue lack may require a cross lip flap operation at primary repair. Before the nose could be brought into exact symmetry it is essential in the early part of the operation to do the following steps of dissection :—

1. The alar attachment to the maxilla must be separated when the lip is separated from the underdeveloped maxilla. The periosteum of the maxilla is not disturbed.

2. On the medial side, the columella is separated from the septum. Then the septal resection is performed so as to place it in the midline proper. A displaced nasal spine may require repositioning, the septum may have to be detached from the vomer and also caudally from the maxilla whilst a strip of cartilage may require excision from its body to bring it to a central position. A triangular part of the septum near its columella attachment has to be removed to increase the columella lip angle and make the nose shorter. In young children this part of the operation should be done on the conservative side, although to quote Sir Harold Gillis " the danger of affecting cartilage growth by early surgery seems overshadowed by the fact that deformed cartilage without correction will continue to grow deformed. On the whole nasal correction is easier when the child is older."

3. Next both the alar cartilages are undermined from the overlying skin from the region of the alar base by an intercartilaginous or by a paramarginal incision in the nostril.

The next step is to form the floor of the nostril so that it is equal in breadth to the normal side. After this it is advisable to judge the nostril openings.

The alar margin must be at the same level at both sides. Good repair produces the tubing of the alar cartilage so that its inner surface is now pointing towards the columella and at the same time the columella becomes straight.

The following defects may still persist and should be carefully noticed at this stage as each of these defects will have to be appropriately corrected.

1. Unequal half dome or tip of the nose. As pointed out before, since the medial crus of the alar cartilage is shorter than the normal side, the rational method of treating this condition is to divide the medial crus in its middle and raise the half-dome of the nose to the level of the opposite side and keep it there with a suture to the opposite cartilage. An external incision could be avoided if the septum is separated from the columella as

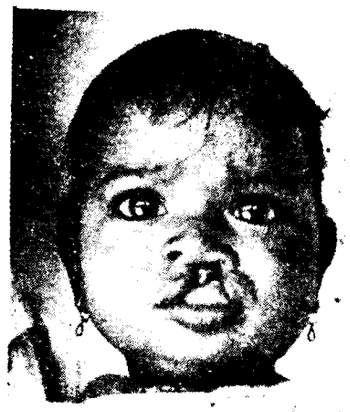

Fig. 7 Before.

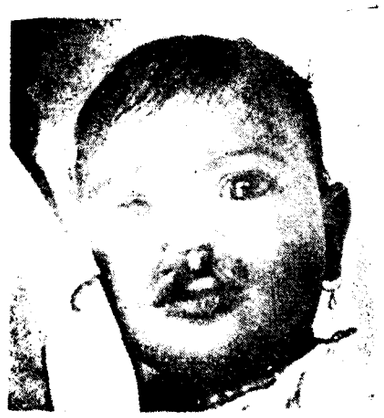

Fig. 8 Before.

Fig. 9 After.

Fig. 10 After

Figs. 7, 8, 9 & 10: Unilateral complete Harelip. Lips repaired by Hagedorn technique.

Fig. 11 Before.

Fig. 12 After.

Fig. 13 After.

Figs. 11, 12 & 13: Unilateral Harelip. Fig. 12 Slight notching of the lower alar cartilage requiring a Z plasty and may be a small cartilage graft. One of the earlier cases of the series. Fig. 13 shows fairly good nasal floor on both sides. Notching of lip margin requires Z plasty operation.

described before and the crus elevated. Humby has suggested rotating part of the normal lateral crus of the alar cartilage to build up the depressed tip of the nose, whilst even lowering the normal side of the tip may serve the same purpose but will shorten the height of the nose. The depressed side of the nose can be lifted by a free cartilaginous implant from the ear.

2. Depression or notching of the lateral crura of the alar cartilage. This outstanding defect may persist in spite of thorough undermining of the cartilage from the overlying skin. In an infant as the cartilage has not lost its spring and when adequately freed, assumes a natural shape similar to that on the normal side. If it persists it has been corrected by the author as follows :

(*a*) Z plasty in the fold or depression. Two cartilage and mucosa bearing flaps are rotated, the central limb of the Z plasty being along the depression. This alone is sometimes effective in total correction of this defect.

(*b*) The depression can be filled up with a cartilage graft through a paramarginal incision in the mucosa. (All pieces of cartilage removed during the operation are carefully preserved by the sister in moist gauze and not thrown away till the end of the operation). The author has often combined the above two methods and succeeded in correcting the depression of the nostril.

3. The lower alar margin on the affected nostril has a certain amount of droop. It can be corrected by an elliptical excision of skin, cartilage and mucosa from the margin of the nostril ; unfortunately this leaves behind a scarred area at the nostril margin and is hence best avoided. Another method is to excise an ellipse of cartilage and mucosa at a higher level and the gap is closed thus reducing the amount of droop of the nostril. Barsky keeps this strip of cartilage attached medially which is then swung upwards and sutured to the cartilaginous dorsum thus correcting the droop. This elliptical excision can be combined in the Z plasty operation described previously when the medial flap of the Z plasty is excised partially, thus producing a combined effect on the droop as well as on the notch in the cartilage.

4. A web of mucosa in the lateral half of the nostril. This is corrected either by a small Z plasty or by a V-Y advancement.

5. Hump and asymmetrical bridge of the nose. This part of the operation should be conservatively done in younger people but is essential in an adult patient undergoing a primary operation. The fact remains that to do justice in a hare-lip repair, the surgeon should be adept in the operation of corrective rhinoplasty. The nasal hump is reduced, the upper lateral cartilage requires shortening and the nasal bones may require to be placed in the proper position after a nasal infracture.

6. If in any case there is a lack of septal support with the original cleft or the support has been lost due to infection, trauma or septal operations,—*in all such instances it is worth while inserting an L shaped bone or cartilage graft with great benefit to the appearance of* the patient.

7. If the lower jaw is protruding giving rise to the appearance of prognathism then a recessive ramisection is indicated.

Secondary hare-lip corrections

The number of patients requiring secondary hare-lip repair is an index of badly done hare-lip surgery. Each individual defect has to be studied preoperatively and the operation planned out. Holdsworth states "accurate and correct diagnoses is essential before formulating the plan of treatment in secondary hare-lip repair. Failing this, operations will be performed unnecessarily, each mistaken endeavour subtracting tissue which is valuable and irreplaceable. The pursuit of perfection however praiseworthy should have a limit and the tendency to keep on trying to achieve by surgery an illusive result must be arrested at some point, unless the patient is to become by vocation a hospital inmate."

The defects which were stated in the primary repair are the ones that usually persist if the technique has been defective. The correction of each of those is already mentioned and need not be repeated. Defects of the lip itself are usual accompaniments of a defective nose, and are beyond the scope of this article. The operation usually means a completely free dissection of all the parts and resetting the whole nose and lip—a major procedure but with very gratifying results. It may be anticipated that with the passage of time the necessity for secondary procedures will decrease as the technique of primary repair is perfected. The following defects not mentioned previously may require correction :

1. Wide nostril floor. Diamond or oval excision of the nostril floor and bringing the edges together corrects this defect.

Fig. 14 Before.

Fig. 15 After.

Figs. 14 & 15: Secondary Harelip. Drooping of the alar margin corrected by excision of excess at the intercartilaginous level. Lip scars revised.

Fig. 16 Before.

Fig. 17 After.

Figs. 16 & 17: Secondary Bilateral Harelip. Treated primarily elsewhere. Note very tight upper lip as the central part has been used for the nose columella. Complete collapse of dental arch.

Fig. 17. After Cross lip Abbe flap operation. Child still requires orthodentic treatment for collapsed front of maxilla.

2. Narrow nostril floor. Here tissue from outside has to supplement the tissue loss. This is done by rotating a flap from the lateral side of the nostril or reducing the nostril floor of the unaffected side.

3. Depressed nostril floor. This is corrected by rotating soft tissue from the cheek around and under the nostril base and floor, by free grafting the nostril floor with dermo-fat or bone.

Double Hare-lip

In a double hare-lip there is shortage of tissue on both sides. The columella is very short and often there is lack of septal support. It is sometimes very difficult to decide in a primary repair of a double hare-lip whether the soft tissue of the premaxilla should be used for the lip or utilised for the columella. Hence there is a constant tug-of-war between the columella and the lip for this small amount of tissue. If the columella is lengthened the nose has a chance to grow properly but the upper lip is very often tightly stretched. An ideal answer to this solution is to borrow tissue from the lower lip which has an appropriate amount of excess and is ready to deliver it. This is done by Abbe's operation in which a triangular segment of the lower lip is rotated into the defect in the upper lip and completed in two stages. The nostrils in a double hare-lip have a porcine appearance and this can be corrected by a triangular excision near the alar base. If septal support is missing then an L-shaped implant is necessary. This can also be performed in a young child so as to allow free development of the nose and when the patient has reached maturity a second and bigger implant may be used. If the lower jaw is protruding a recessive ramisection will restore the facial contour.

Thought for the future: Amongst the congenital defects, Fogh Anderson noted in a large series that harelip and cleft palate defects have a much higher incidence than other conditions like congenital hearts. Although the ideal is to have special Hare-lip and Cleft Palate Centres where these defects can be treated like the Lancaster Cleft Palate Clinic and the Allurtown Hospital Clinic in Penna, in U.S.A., a beginning should be made to have a plastic unit in each of the teaching hospitals of India. The number of cases requiring plastic reconstructions is so great that they can keep several plastic units busy for all the days of the week in each major town of India! Sir H. Gillies recently stressed the need for a plastic unit in each teaching hospital. Absence of a speech therapist makes cleft palate repair an anatomical surgical exercise, absence of a good orthodentist in each hospital prevents the healthy and productive teamwork so essential for a good hare-lip result.

Conclusion: I have put down my experience of 41 cases of harelip repair. I can claim to know all the questions and difficulties but I do not claim to know all the answers. This paper is written, so that it would produce an interesting discussion about this subject which has always intrigued me. The fascinating thing about this surgery is that there are several answers to the same problem and all aiming at the same result. To conclude, it reminds me what Dr. Millard has said in his excellent paper read at the First International Congress of Plastic Surgery in Stockholm in 1955 " It has been said that a natural looking result following closure of a congenital cleft is a work of art. In fact it is a 3-D work of sculptured art. Principles, measurements, marks and incisions of a technique can be standardised and a blueprint of the technique memorised. Yet the last few millimeters which make all the difference must depend upon the *sculptor* and his *clay*.

My thanks are due to Dr. R. G. Talwalkar, F.R.C.S., Hon. Surgeon, G.T. Hospital where the majority of the cases have been operated on and to Dr. P. Prakash B.D.S.,M.S. (U.S.A.) Orthodentist for useful co-operation.

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THE LEFT LATERAL POSITION FOR ELICITATION OF APPENDICULAR TENDERNESS

BY

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Any written account about appendicitis generally includes a reference to the McBurney point as an important diagnostic physical sign. Some neuroanatomists argue that this point remains constant regardless of the exact location of the appendix. Experience teaches however that the maximum tenderness in any given case of appendicitis is located wherever the involved appendix happens to be. When the inflamed appendix happens to lie in the pelvic cavity, maximum tenderness will only be elicited during a digital examination through the anal canal. In a case of retrocoecal appendicitis the tender spot is found along the lateral border of the right Erector Spinae muscle. The position of the tender spot will vary along this line according to the location of the appendix. In a very high placed appendix the tender spot may be as high as the renal angle. In all other cases the maximum tender spot in appendicitis is in relation to the anterior abdominal wall in the right iliac fossa but its position may vary. Rarely it is as high as the gall-bladder.

The McBurney point has been described differently in different text-books of surgery. In these descriptions no consideration is given to the contour and condition of the abdominal wall. It is assumed in some quarters that the caeco-appendicular junction is a fixed point. It was therefore not surprising to see an examiner in surgery asking the candidates to give the exact surface marking for the caeco-appendicular junction, and woe to the candidate who failed to demonstrate the surface marking as the examiner required!

In Nelson's loose leaf surgery the McBurney point is defined as either the mid-point of a line joining the anterior superior iliac spine to the umbilicus or the intersection of that line and the lateral margin of the right rectus muscle. However the writer of that article rightly points out that the maximum tender spot in appendicitis is not constant and proceeds to describe two other points. If the most tender spot happens to be a little lower than the intersection of the two lines just described, it is known as Clado's point. If the tender spot is two inches lower than the mid-point of the spino-umbilical line it is known as Lotheissen's point.

It is interesting to observe that in his original writing published in 1889 Charles McBurney said that the maximum tenderness would be ascertained in the *average adult* by the pressure of *a single finger tip* and this point was exactly two inches from the anterior iliac spine on a line drawn from this process to the umbilicus. This teaching is often forgotten. To emphasise this teaching the late Sir Holburt Waring of St. Barts Hospital used to ask the patient himself to touch with the tip of the index finger the different points of his abdomen and indicate which he considered to be the most tender spot. Thus he impressed upon his students the importance of eliciting appendicular tenderness with the moderate pressure of a single finger tip. One therefore shudders when one sees some wholesale appendicetomy merchants push down vigorously with four fingers into the right iliac fossa until the patient winces under the pain induced thereby.

It is not possible to elicit such finger tip tenderness in an obese patient when he is examined in the usual supine position and particularly when the appendix lies deep, being overlapped in succession by the caecum, coils of small intestine, a fat-laden omentum and a varying thickness of the abdominal wall. In such and in fact in all cases of suspected appendicitis it will be a distinct advantage to examine the patient in the left lateral position in addition to the routine supine position.

Ask the patient to take either a half or a full turn to the left with the legs drawn up. It will be noticed that the heavy abdominal wall falls away to the left and a relatively fat free part of the abdominal wall comes to lie over the right iliac fossa. The intra-abdominal structures overlying the appendix also fall over to the left. The appendix thus comes to lie close to the abdominal wall. Further it is surprising to find in a majority of cases, that the abdominal wall covering the right iliac fossa feels considerably relaxed in this lateral position. It is therefore possible to elicit

appendicular tenderness with gentle pressure. One finger tip palpation shows that the maximum tenderness may lie anywhere within a short distance of the point originally described by McBurney.

When an appendicetomy is done with the patient in a modified left lateral position it is observed on opening the abdomen with a gridiron incision that the structures overlying the appendix fall over to the left and the appendix is easily identified. Those surgeons who do their appendectomies in this lateral position will easily appreciate the value of the lateral position in eliciting appendicular tenderness.

In a skiagraphic study of the appendicular region a full or a semi-left lateral position will be a great help in delineating the appendix by causing all overlying shadows to fall out of the way. A series of films from a recent study of a case by my young colleague and radiologist, Dr. J. Sidhwa, has demonstrated the point under consideration.

For the last twelve years appendicular tenderness has been looked for, both in the supine and the left lateral positions. The resulting clinical experience teaches that the left lateral position has decidedly helped to establish the diagnosis in almost all doubtful cases. Further it has helped to determine the level at which the incision is to be placed for removal of the offending appendix.

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CARCINOMA OF THE THYROID

(*A Clinical Study of* 108 *cases*)

BY

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Malignant neoplasms of the thyroid gland exhibit marked differences in morphology and hence there is a great deal of variation in the clinical course of the disease. This is well illustrated in the following two cases :

T.M.H. Case No. 1693 : A 35 year old Muslim male visited the hospital for the first time on 3-1-1942 with history of gradually enlarging painless lumps on the right side of the neck which were noticed by him almost five years before. Physical examination revealed a tiny hard nodule in the right lobe of the thyroid and multiple lumps on the right side of the neck. Some of these lumps were hard to feel, whilst others were cystic in consistency. A biopsy from one of the lumps suggested metastases from a carcinoma of the thyroid. A total thyroidectomy with a radical neck dissection was done on 9-1-1942. The report from the Department of Pathology was a Papillary Carcinoma of the thyroid with metastases to the cervical lymph nodes. Six months later he came back with enlarged lymph nodes on the left side of the neck. A radical neck dissection was done on the left side on 4-6-1942. Examination of the lymph nodes showed the same type of disease—Papillary Carcinoma. The patient has been followed up regularly till 1956 without any evidence of recurrence.

T.M.H. Case No. 6321 : A Hindu female, 50 years of age, was first seen at the hospital on 16-2-1944, complaining of a swelling of five months duration in the region of the thyroid gland. Examination revealed a hard solitary mass in the right lobe of the thyroid. There were a few small palpable nodes in the neck on the right side. Lungs were reported clear, but her blood pressure was raised. After preliminary preparations she was operated upon. The entire affected lobe of the thyroid and the isthmus were excised and a radical neck dissection was performed at the same time. The report from the Department of Pathology was "A Giant Cell Carcinoma of the thyroid with metastases to the lymph nodes." Three months later she came with history of cough and fever and an X-ray picture of the chest revealed extensive bilateral pulmonary metastases and finally she expired within five months of her first visit to the hospital.

Judging from the records of cases at the Tata Memorial Hospital, carcinoma of the thyroid appears to be an infrequent disease. During a period of 16 years (1941–1956), there were only 108 cases with definite histological evidence of the disease, from a total of 32,058 cases of carcinoma, giving an incidence of only 0.33 per cent. Pathological conditions of the thyroid are more often seen in the female sex and so is cancer. In our series however, there was a preponderance of the disease in the males. Could it be that our women are still reluctant to receive hospital treatment ?

Table I indicates that almost 75 per cent of cases of carcinoma occurred between the age of 40 and 70 years and that when the disease occurred in the younger age group, the histological variety was mostly a papillary carcinoma. Contrary to the usual belief, the anaplastic variety occurred more often in the older age group. In our series the youngest subject was a male child of two years and the oldest was a male of 78 years. Carcinoma seldom occurs in a person suffering from a toxic goitre. There was just one case in our series who had a carcinoma associated with symptoms of thyrotoxicosis. The relationship between a non-toxic nodular goitre and carcinoma is very striking. It is reported [1, 3, 4] that the incidence of carcinoma in multinodular goitres varies from 2 to 10 per cent, whereas the incidence is almost 20 per cent in thyroids which present a solitary nodule. Some observers believe that the so-called solitary nodule may be a slow growing carcinoma from the start. Gault and Thomas[2] have reported the incidence of carcinoma as 25.8 per cent of all nodular goitres, and in our own series it is 21 per cent. Carcinoma has been observed to occur with greater frequency in nodular goitres of children and young subjects under the age of 15 years. Winship and Chase[5] have reported an incidence of 29 per cent.

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TABLE I

Analysis of 108 Cases according to the Age, Sex and Histological variety

Age in years	Papillary Ca.			Follicular Ca.			Anaplastic			Hurthle			Epidermoid			Grand total
	M	F	Total	M	F	Total	M	F	Total	M	F	Total	M	F	Total	
1–10	...	1	...	1	...	...	...	...	...	...	...	...	...	...	...	1
11–20	...	2	...	2	...	...	...	...	...	...	...	...	...	...	...	2
21–30	...	4	1	5	2	2	4	...	...	...	1	...	1	...	...	...	10
31–40	...	4	3	7	3	1	4	1	1	2	...	1	1	...	...	...	14
41–50	...	4	7	11	6	9	15	4	2	6	...	1	1	1	...	1	34
51–60	...	4	4	8	5	6	11	4	2	6	...	...	...	2	...	2	27
61–70	...	1	1	2	7	3	10	3	1	4	...	...	...	...	...	...	16
71–80	...	1	1	2	2	...	2	...	...	...	...	...	...	...	...	...	4
		—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Total	...	21	17	38	25	21	46	12	6	18	1	2	3	3	...	3	108

Malignant tumours of the thyroid gland are mainly adenocarcinomata but it is preferred to divide them into two main groups—the differentiated and the undifferentiated—and these in turn are sub-divided according to the cell type which predominates. Such a classification not only describes the histological appearance of the tumour but also correlates the clinical course of the disease and aids in the selection of therapy and indicates the prognosis. In the differentiated group are the papillary and the follicular carcinomata. In a large series of cases the papillary variety is found to be more common. It is less malignant in nature and is often seen in the younger age group. Calcification in the tumour mass of its metastases is a special feature. Cervical metastases from a papillary carcinoma in a young subject have been occasionally mistaken for tubercular lymphadenitis, particularly when the primary in the thyroid is small and there is evidence of calcification. Prognosis is quite favourable in papillary carcinoma. Follicular carcinoma is less common and spreads more often via the blood stream to distant organs. Prognosis is less favourable in this variety.

The undifferentiated or the anaplastic group includes the small cell and the giant cell carcinomata. Their main features are that they occur with greater frequency in the older age group and are rapid in local and distant spread : in spite of radical surgery recurrences are frequent, hence there is a tendency to rely on radiotherapy. Epidermoid carcinoma of the thyroid is a very rare condition and so is Hurthle cell carcinoma.

The management of a case of carcinoma of the thyroid depends upon the clinical state in which the disease presents itself.

Clinically evident cancer : When a case presents a large hard fixed mass in the region of the thyroid gland, perhaps with paralysis of the vocal cord and cervical lymph node metastases, the chances of cure are extremely poor as there is no specific treatment except to relieve the pressure over the trachea. When the disease is moderate in extent, an attempt is always made to remove as much of the growth, as possible and the residual disease is treated either with deep X-radiation or with radon gold seeds inserted locally. Such a procedure is well worth trial particularly in a rapidly growing variety of carcinoma. In our series, 24 cases had this type of treatment with the result that 8 of them had no clinical evidence of disease at the end of five years.

In an operable case, a total thyroidectomy is considered the treatment of choice, but some authorities are satisfied with a complete excision of the affected lobe along with the isthmus and resort to a total thyroidectomy only if there is suspicion of the disease creeping to the other lobe. When there is clinical evidence of cervical metastases, a radical neck dissection should always be done simultaneously on the

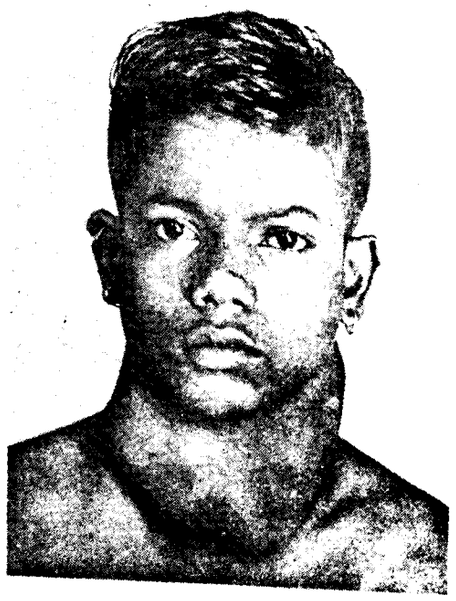

Fig. 1
A papillary carcinoma of the thyroid in a lad of 12 years.

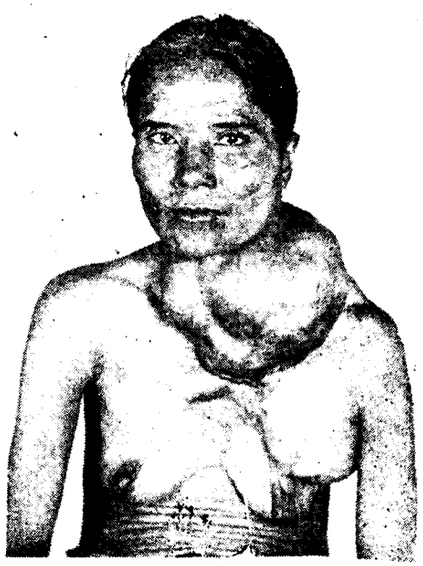

Fig. 2
An advanced case of carcinoma of the thyroid.

side of the maximum disease. Very seldom an occasion arises for a bilateral neck dissection. Radical surgical procedures were possible in 11 cases in our series.

As in other malignant conditions in the region of the head, face and neck, so in cancer of the thyroid, the question of a prophylactic neck dissection always comes up for discussion. We recommend a prophylactic neck dissection when we are reasonably sure that the patient will not come for regular follow-up examinations. Secondly a prophylactic neck dissection is justified in the case of papillary carcinoma of the thyroid which invariably metastasises to the cervical lymph nodes and being a slow growing tumour offers the best chance for cure.

Though radiotherapy alone is not the best treatment for cancer of the thyroid, yet it is always recommended for cases who refuse surgery, particularly in elderly persons who have an anaplastic variety of tumour. In our series, there were 25 cases who received this therapy. The disease remained controlled in 7 cases at the end of three years and of these three were well and alive at the end of five years.

Clinically suspected cancer : The possibility of cancer is always borne in mind whilst treating a case of non-toxic nodular goitre, particularly when the mass is unilateral. The nodule is excised with a liberal amount of thyroid tissue all around. It is cut open and inspected while the patient is still on the operation table. At this stage a frozen section diagnosis, if available, could be of immense value, because a definite diagnosis of cancer obtained at the time of operation helps the surgeon to plan a radical procedure best suited to the case. Nodular enlargements of thyroid in children often turn out to be malignant in nature and, therefore, it would be a wise policy to perform radical excisions in them.

Occasionally, a nodule which has been excised on the clinical impression of an adenoma of the thyroid, is reported a few days later by the Pathologist to be a carcinoma. What should be the line of treatment in such a case ? Should the case be observed, reoperated on or radiated ? Of course the choice of treatment will depend upon the judgment and experience of the person treating the case, but the following points are worth considering. In a young subject with a differentiated variety of carcinoma, the best procedure would be to offer radical surgery ; but in an old person with an undifferentiated type of carcinoma, preference may be given to radiotherapy.

Rarely, a carcinoma of the thyroid is diagnosed for the first time after it has metastasised to a distant place. A careful examination then reveals either a tiny hard nodule in the thyroid or a longstanding non-toxic nodular goitre. These are the cases which could have been salvaged by early surgical intervention for their thyroid enlargements. The following case is worth reporting. T.M.H. Case No. P-2385 :—A Hindu female of 55 years was first examined in the Out-Patient Department on 18-5-1954 with the complaint of a painful swelling in the left side of the mandible. The swelling had been gradually increasing in size for the past four months. Examination revealed a firm swelling which had expanded the horizontal ramus of the left side of the mandible in the region of the molars. There was no ulceration of the mucous membrane. No other abnormality was detected at the time of the examination in the O.P.D. An X-ray picture showed an osteolytic but loculated lesion which had expanded the bone. The radiological diagnosis was "most probably a malignant giant cell tumour." A biopsy through a thick bore needle revealed the nature of the disease to be a metastatic deposit from a thyroid carcinoma. A very careful examination now revealed a tiny hard nodule in the left lobe of the thyroid, about which the patient had no knowledge. It was extremely hard to convince the patient that the disease in the mandible was due to the nodule in the thyroid and she refused any treatment to the thyroid and accepted only radiotherapy for the metastases. Three years later, she was seen with the disease in the mandible much smaller in size, but now had pulmonary metastases. Curiously, the nodule in the thyroid was just the same in size and there were no metastases in the neck.

When a malignant change occurs in an intrathoracic goitre the case may present interesting clinical features. The following case is cited. T.M.H. Case No. M-1508: A Hindu male of 47 years was examined on 23-4-52 with a complaint of cough and loss of weight of two months' duration. Except a few enlarged veins in the neck and slight dullness over the

Fig. 3
T.M.H. Case No. P. 2385. An X-Ray picture shows an osteolytic lesion in the mandible.

Fig. 4
T.M.H. Case No. 2385. A small nodule as seen in the left lobe of the thyroid.

superior mediastinum, physical examination revealed no other abnormality. X-ray picture of the chest showed a lobulated swelling in the superior mediastinum reported as "probably enlarged lymph nodes." With these findings a provisional diagnosis of a lymphosarcoma of the mediastinal lymph nodes was made. He was placed on a trial therapy with deep X-rays, which was soon stopped because it showed no improvement in the size of the mass. A few weeks later the man came with enlarged lymph nodes in the left supraclavicular region. A node was excised and was reported to be a metastatic deposit from a carcinoma of the thyroid gland. Further examination failed to demonstrate any connection with the thyroid gland in the neck.

Radioactive Iodine (I-131) has proved useful in a limited number of cases of carcinoma of the thyroid ; only 15 per cent of carcinoma pick up I-131 in effective concentration. Sometimes, distant metastases in the bones are made to take up Radioactive Iodine after a wide excision of all the thyroid tissue.

Prognosis of carcinoma of the thyroid depends on several factors. By far the most important is the stage of the disease. Prognosis is best in a case where the disease is suspected or has been discovered accidently in a thyroid nodule ; but when the disease is clinically evident, the prognosis is less favourable, particularly when there is extension to the cervical lymph nodes. Papillary carcinoma is much more frequent and because of its less aggressive nature and restricted spread to the cervical lymph nodes, the prognosis in this variety is much better than in other histological varieties. Carcinoma in the younger age group has a better prognosis because of the frequency of the papillary variety and because young subjects are better suited for radical surgical procedures. Since surgery is the treatment of choice for carcinoma of the thyroid, the prognosis will vary according to the extent of the radical procedure. Finally when surgery has been unsuccessful or has been refused, the prognosis will be influenced by the radiosensitivity of the tumour.

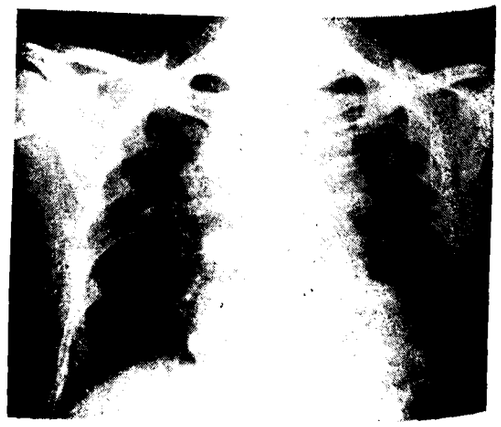

Fig. 5
T.M.H. Case No. M. 1508. An X-Ray picture showing an intra-thoracic carcinoma of the thyroid.

Fig. 6
Microphotograph showing an epidermoid variety of carcinoma of the thyroid.

Fig. 7
Microphotograph showing "Hurthle Cell" carcinoma of the thyroid.

Fig. 9
Microphotograph showing a Follicular variety of carcinoma of the thyroid.

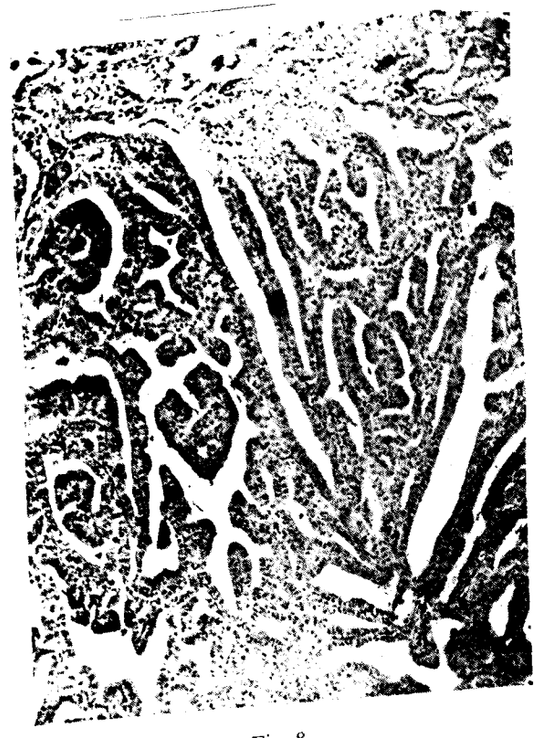

Fig. 8
Microphotograph showing a Papillary variety of carcinoma of the thyroid.

Fig. 10
Microphotograph showing a spindle cell variety of carcinoma of the thyroid

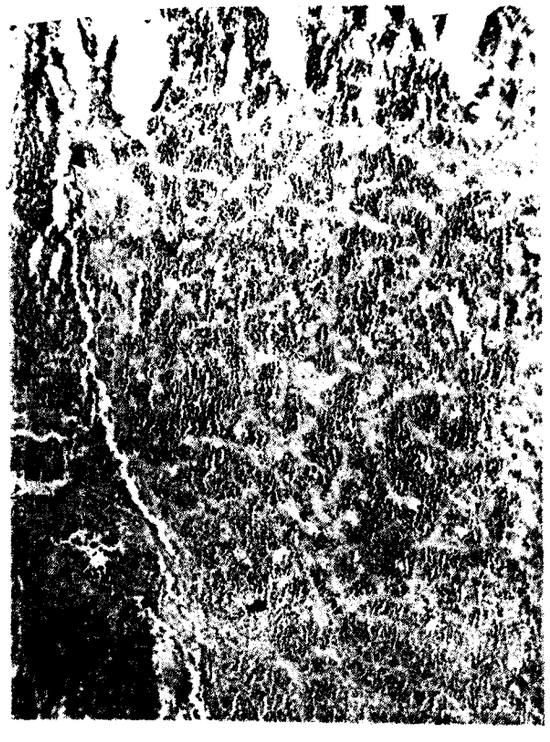

Fig. 11
Microphotograph showing a small round cell variety of carcinoma of the thyroid.

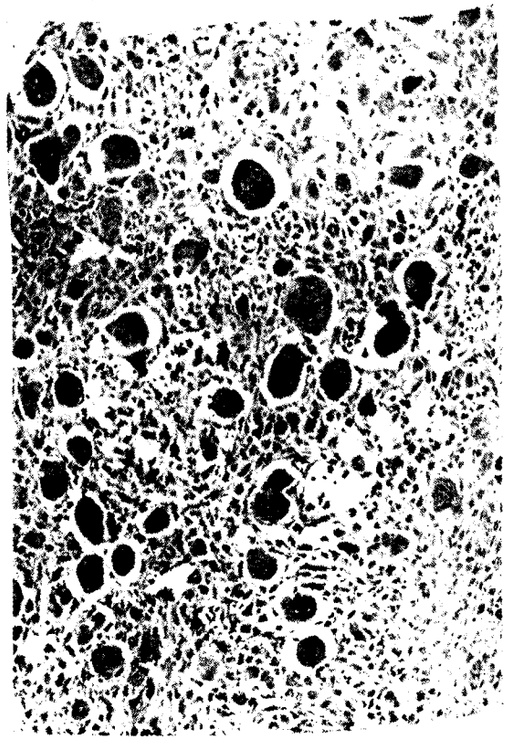

Fig. 12
Microphotograph showing a giant cell variety of carcinoma of the thyroid.

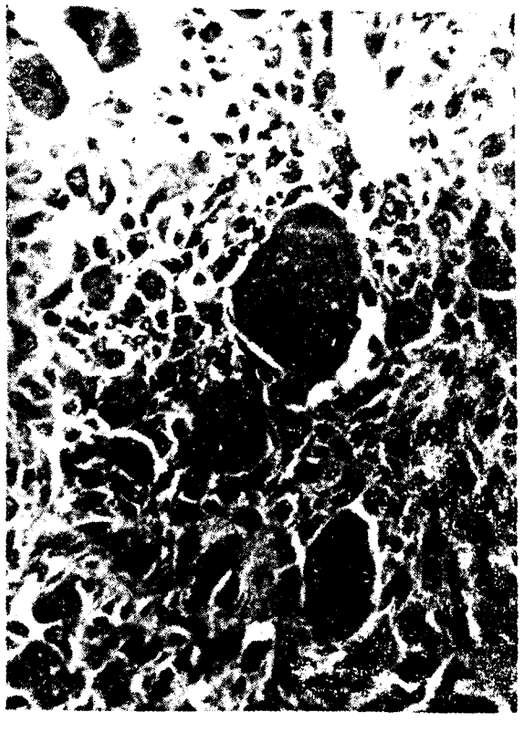

Fig. 13
Same as fig. 12. (High power)

Summary

1. Incidence of carcinoma of the thyroid in the recorded cases at the T.M.H. is 0.33 per cent.

2. Incidentally, in our series of 108 cases, the disease occurred more often in males and the follicular variety was slightly more common than the other histological varieties.

3. 10 per cent of cases in our series could be subjected to radical surgical treatment and those in whom post-operative radiotherapy had to be given, nearly one-third of them survived at the end of five years.

4. Radiotherapy is worth trial in those who refuse surgery or who cannot be subjected to surgical procedures.

5. Radio-active Iodine where available, has proved to be a valuable adjunct in the treatment of carcinoma of the thyroid.

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RESECTION IN PULMONARY TUBERCULOSIS

(Experience in 75 cases)

BY

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In this paper we review 78 consecutive resection procedures performed for pulmonary tuberculosis on 75 patients at the Thoracic Surgical Centre, Silver Jubilee Tuberculosis Hospital, Delhi since its inception, and followed up from 6 months to two and a half years. The Centre was started by a W.H.O. team in April, 1954, but due to certain difficulties, some of which are still to be overcome, resection work could not be taken in hand before February, 1955.

TABLE 1

(Age and Sex)

Age Group	Male	Female	Total
All ages	46	29	75
Under 10	1	...	1
10—19	6	10	16
20—29	20	15	35
30—39	12	2	14
40—49	7	1	8
50 and over	...	1	1

Age and sex

Of the total 75 cases there were 46 males and 29 females, a ratio of 3 : 2. The age groups at the time of operation, and the sex, are given in table I. The youngest patient was 9, and the oldest 50 years of age, 35 out of 75, or about 50%, were in the 20–29 year age group.

Indications

The type of parenchymal disease indicating resection is shown in table 2. In 3 cases there were double indications. In 2 of these there was a solid focus associated with a cavity, while in the third a right upper lobe cavity co-existed with stenosis of the right lower lobe bronchus. 28 of the 75 cases had destroyed lung or lobe,

TABLE 2

(Indications)

Type of lesion	Male	Female	Total
Destroyed Lung :			
Right ...	3	1	4
Left ...	3	8	11 — 15
Destroyed Lobe :			
R.U.L. ...	4	3	7
R.U. and M.L. ...	...	1	1
L.U.L. ...	4	1	5 — 13
Cavities :			
Right Lung ...	2	...	2
Left Lung ...	2	...	2
R.U.L. ...	3	8	11
L.U.L. ...	7	5	12
Apical —R.L.L. ...	2	...	2
Basal —R.L.L. ...	2	...	2 — 31
Bronchiectasis :			
R.U.L. ...	1	...	1
R.L.L. ...	1	...	1
R.M. and L.L. ...	2	...	2
L.U.L. ...	1	...	1
Ap-pst R.U.L. ...	1	...	1
Ap-pst L.U.L. ...	1	...	1 — 7
Solid Focus :			
Apical —R.U.L. ...	1	...	1
Ap-pst R.U.L. ...	1	...	1
Ap-pst L.U.L. ...	1	...	2
with R.U.L. cavity ...	2	...	— 5
Broncho-stenosis :			
R.L.L. ...	...	1	1 — 1
Failed Thoracoplasty :			
Right ...	3	2	5
Left ...	1	...	1 — 6
All ...	48	30	78

and the incidence was considerably higher in the females, being 47% as against 29% in the males. The incidence of cavities was also slightly higher in the females, being 45% as against 40% in males. There was no bronchiectasis in the females, and no woman had disease restricted to a segment. This analysis suggests that more women suffer from the more severe forms of the disease. This we feel can be explained by the prevailing socio-economic conditions in this country. Among the poor uneducated folk the male, being the actual or prospective bread winner and the privileged sex, gets priority and better treatment.

TABLE 3

(Pre-operative Duration of Disease)

Duration in Years	Male	Female	Total
All cases	46	29	75
Upto 1 year	...	...	...
Over 1 and upto 2 years ...	15	11	26
Over 2 and upto 3 years ...	8	11	19
Over 3 and upto 4 years ...	13	3	16
Over 4 and upto 5 years ...	5	1	6
Over 5 years	5	3	8

Material

It is impressive that all the patients had had prolonged treatment which failed to arrest the disease. The duration of the disease before operation is shown in table 3. No patient came up for surgery within one year of the known commencement of disease; and 26, or one-third, came up within 2 years, and 30, or 40%, had been suffering for more than 3 years at the time of operation. The condition of the residual lung tissue is equally impressive. Only 18 out of 75, or 25%, showed no disease in the residual lung on plain skiagraphy, although of the remaining 57 in only 2 was it regarded unstable at the time of surgery. In one of these the right upper lobe was resected as an emergency due to recurrent hemoptysis during her convalescence from infective hepatitis. In the other a small right upper lobe cavity kept appearing and disappearing while she waited to have a destroyed left lung removed.

Then again, of the total 75 only 38 cases, or 50%, had negative sputum pre-operatively, and only 4 came up for surgery within the original continuous antimicrobial treatment, whereas 71 had been exhibited multiple interrupted courses by the time they reached us. 17 of the 31 cavity cases, or 55%, and 3 of the 6 thoracoplasty failures, were open positive. We regret we have no facilities available yet to determine the sensitivity of the tubercle bacillus.

TABLE 4

(Operations)

Operation	Male	Female	Total
Pleuro-pneumo :			
Right ...	1	...	1
Left ...	...	1	1 — 2
Pneumonectomy :			
Right ...	3	2	5
Left ...	8	10	18 — 23
Lobectomy :			
R.U.L. ...	12	12	24
R.U. and M.L. ...	2	2	4
R.M. and L.L. ...	2	...	2
R.L.L. ...	3	...	3
L.U.L. ...	9	3	12 — 45
Segmental Resections :			
Apical — R.U.L. ...	1	...	1
Ap-pst — R.U.L. ...	1	...	1
Apical — R.L.L. ...	2	...	2
Ap-pst — R.U.L. ...	...	...	...
Subseg ... R.M.L. ...	1	...	1
Ap-pst — L.U.L. ...	2	...	2
Ap-pst ... L.U.L. ...	...	...	...
Apical + — L.L.L. ...	1	...	1
All Types ...	48	30	78

Surgical procedures and routine

The 78 surgical procedures performed are listed in table 4. There were 25 pneumonectomies including 2 pleuro-pneumonectomies, 45 lobectomies, and 8 segmental resections. 4 upper lobectomies had apical segments of the lower lobes also removed with them. Among the 78 procedures 4 were emergencies—one for a recurrent hemoptysis from a destroyed right upper lobe, and 3 for uncontrolled toxaemia due to a destroyed left lung. Of the 29 females none came up for a segmental resection, while of the 25 pneumonectomies 13 were performed in 29 females and 12 in 46 males. The ratio of Lobectomies is about equal in the 2 sexes. It is thus seen again that more females had the more serious operations performed on them.

3 patients had 2 resections each, the second operation having been performed to treat the bronchopleural fistula. In 2 a left upper lobectomy had to be extended to a pneumonectomy, and in the third the apical segmental resection was converted to a right upper lobectomy.

We operate in the lateral position, and use diathermy throughout. A post-resection space-reducing thoracoplasty is done in every case except the pure segmental resections. Harley has stressed the potential danger of any reversible lesions in the residual lung tissue reactivating due to overdistension. Bickford, Edwards and associates reported 10 cases in whom foci in the left lower lobe apex reactivated after upper lobectomy without space-reducing thoracoplasty. It occurred in no case when corrective thoracoplasty was carried out. Post-operatively the patient is kept in the hospital for 6 months atleast, and throughout this period, as also for 2 months at home, he is under continuous antimicrobial cover. As regards the immediate preoperative therapy it has not been possible, due to certain reasons beyond our control, to follow a set routine. Of the 75 cases in only 35 was it possible to start the antimicrobials a month or 3 weeks before the operation.

Respiratory function estimations including broncho-spirometry, and histopathological and culture studies of the resected specimens were and are made available to us through the kind co-operation of the Patel Chest Institute.

Surgical mortality

The post-operative deaths are classified in table 5, both case-wise as well as operation-wise.

TABLE 5

(Post-operative Deaths)

Cases	Male	Female	Total
(A) Case-wise :			
First 25		6	6
Second 25		2	2
Third 25		2	2
All Cases		10	10

(B) Operation-wise :

Type of operation	Number	Deaths
Pleuro-pneumo ...	2	1
Pneumonectomy ...	23	4
Lobectomy : ...	45	5
Segmental resection ...	8	...

TABLE 6

(Causes of Death)

1.	Cardiac Arrest	2
2.	Cerebral Anoxia	2
3.	Shock	3
4.	Vascular accident	1
5.	Pulmonary embolism	1
6.	Transfusion reaction	1
	All causes ...	10

10 patients, all females, died within 6 weeks of surgery, an arbitrary period most commonly used in computing operative mortality. 6 of the 10 deaths occurred in the first 25 cases, while in the next 50 cases there were 4 deaths. The causes of death, listed in table 6, are as considered most probable at retrospective discussion in the absence of post-mortem, which was permitted in no case. 2 patients died of cardiac arrest, the catastrophe in both occurring when, after the operation, the patient had been dressed and was ready to go to the ward. Cardiac massage failed. 2 deaths were due to cerebral anoxia occurring sometime during the operation. One died 2 hours and the other 15 hours after the operation without regaining consciousness. Shock was responsible for 3 deaths, blood, plasmosan, and noradrenalin all failing to reverse it. Vascular accident claimed one death. In one case the cause, from clinical and radiological study, was considered to be pulmonary embolism. One case died of mismatched transfusion.

10 deaths in 78 operations, a mortality of 13%, appears at first glance to be rather a high figure, but a re-appraisal of the type of cases we have had to deal with would show that it does not compare at all badly with the figures of Douglass, Bosworth and associates who list destroyed lungs and lobes, cavities more than 1 cm. in diameter and persisting after chemotherapy, tub. empyemata, and thoracoplasty failures as the "Salvage" group, and show a comparably high mortality in this group. Judged accordingly, 72 of our 75 cases fall in the "Salvage" group, and we show a postoperative mortality of 4 in 23 pneumonectomies of 17%, against their 6 in 36 pneumonectomies or 17%. With lobectomies we list 5 deaths in 45 operations or 11%, against their 5 in 57 operations, or 9%.

Late deaths

There have been 6 late deaths in the series, 3 of which were due to a related cause and 3 due to un-related causes. Of the former 2 died of respiratory insufficiency after 2½ and 14½ months respectively, and the third died due to a severe hemoptysis on the 53rd post-operative day. The unrelated causes were infective hepatitis in two cases and an accident in one.

Complications

The frequency and severity of the non-fatal complications are equally important to assess the value of surgery. The post-operative complications are shown in table 7-A.

Vascular accidents : Due to a vascular tear 2 planned left upper lobectomies were converted to pneumonectomy. In the 3rd case the torn pulmonary artery during a right upper lobectomy was controlled, but the patient died due to excessive blood loss.

Thoracic Duct Tear : In one case the thoracic duct was damaged at the post-resection thoracoplasty. The tear was recognised at the table and dealt with by ligation.

TABLE 7-A

Post-operative Complications (within 6 weeks)

Complications	25 Lungs	45 Lobes	8 Segments
Vascular accidents	...	3	...
Bronchiolar leaks	2	1	3
Fistula with Empyema ...	3	1	2
Empyema without Fistula ...	1	2	...
Tub. Spread or reactivation.	1	...	...
Contralateral aspiration lobular collapses	2	3	...
Psychoses	...	1	1
Damage to the Thoracic duct.	...	1	...
Haemothorax	1	...	...

Haemothorax : Excessive bleeding in the left pneumonectomy space occurred in one case. It responded to immediate blood transfusion and later varidase and aspiration.

Simple fistula : This complication occurred in 4 lobectomies and one segmental resection. In one case it responded to re-introduction of the intercostal ctheter and suction, and could therefore be called 2nd degree fistula of Overholt. The other 4 cases were re-thoracotomised. In one the leak was dealt with by direct closure, while in 2 cases the left upper lobe was converted to pneumonectomy, and in one case the apical segment was extended to right upper lobectomy. None of these cases was complicated by an empyema.

Fistula with empyema : This complication occurred in 3 pneumonectomies, 2 lobectomies, and one segmental resection. 2 of these cases died before the fistuval could be tackled surgically —one as a result of severe hemoptysis, and the other due to an accident. This second patient required oxygen at meal times. One day he left the bed to open the oxygen cylinder standing beside, slipped and fell with the cylinder on him. The cylinder was rather heavy and the patient died immediately. No post-mortem was allowed to determine the exact cause of

death. The remaining 4 cases responded to drainage and thoracoplasty.

It is interesting to note that all the 6 fistulas with empyema and 3 of the 5 simple ones, occurred in those patients who had been covered with effective dosage of antimicrobials for 3 weeks or more preoperatively. This is in variance with other reports that show that the incidence of fistula is lower in these cases.

Empyema without fistula : Of the 3 empyemata without fistula 2 responded to drainage and thoracoplasty. The third is still with us, which is actually a case of Samb space infection, since in him thoracoplasty had preceded resection.

Contralateral spread (or reactivation) : Aspiration complication occurred in 6 cases. In every case it took the form of an area of multiple small oapcities in the midzone one the post-operative picture. In 5 cases this area cleared up or left behind only some fibrous streaking, and there was no associated toxaemia or positive sputum. In one case only the area progressed to cavity formation.

Psychoses : 2 patients developed mental symptoms. In one there was a history of previous attacks, and he responded to replacement of I.N.H. with P.A.S. and sedation. In the other patient there was no previous history. He became violent and destructive and had to be removed to Police custody. There all his treatment was stopped, and the intercostal catheter, put in to drain the empyema with fistula, was removed. Surprisingly enough he recovered spontaneously and his fistula closed. He has since been back with us to complete the treatment including the thoracoplasty.

The after-history

57 of the 59 survivals have been followed-up from 6 months to 2½ years. This is only a preliminary report, as 5 to 10 years follow-up is required to give the full story. It has been shown that relapses continue to occur during and after the 10 year period. However, the danger of relapse is greatest during the first 3 years after the disease has been arrested. This follow-up covering upto 2½ years should therefore give an indication of the value of these resection procedures.

The patients who progress satisfactorily are discharged from the hospital 6 months after surgery. But the antimicrobial cover continues for 2 months more at home. After discharge the patient reports regularly at the follow-up clinic every 3 months, when his status is ascertained by clinical, radiological, hematological, and bacteriological examinations as required.

TABLE 7-B

Late Complications (after 6 weeks)

Complication	Time after Surgery	Number
Homo-lateral Spread or re-activation.	10 months after a left upper lobectomy.	1
Contra-lateral Spread or re-activation.	6 months after a right upper lobectomy.	1
Infection of Samb's space.	14 months after a right upper lobectomy.	1
	28 months after a right upper lobectomy.	1

There have been no deaths in the follow-up. The complications are listed in table 7-B. There have been 2 spreads or re-activations. In one case it occurred on the same side—in the left lower lobe apex after the upper lobectomy—10 months after operation and 4 months after discharge. He was immediately hospitalised again and treated with antimicrobials for 6 months. He has now been out again for 4 months and is keeping fit. In the other case the complication occurred in the left midzone 6 months after a right upper lobectomy, and 2 months after a disciplinary premature discharge. He did not continue the antimicrobial cover at home. He is now, re-admitted—and is progressing satisfactorily.

In 2 cases non-tuberculous infection of the post-resection thoracoplasty space occurred 14 and 28 months respectively after the operation. One required admission, and the other was treated as an out-patient.

The overall picture shows that 52 of the 75 cases, or 70%, are in a satisfactory condition 6 months to 2½ years after the resection procedure. This, we believe, compares quite favourably with the figures of Douglass, Bosworth and Associates, who show a 75% salvage in failed thoracoplasties, and a 50% salvage in destroyed lungs.

Summary

1. 78 resections performed in 75 cases have been analysed.

2. There were 10 post-operative and 6 late deaths.

3. 57 of the 59 survivals have been followed up from 6 to 30 months.

4. There were no deaths in the follow-up.

5. There have been 2 spreads or re-activations and 2 infections of the Samb space in the follow-up. All the 4 complications have responded to treatment.

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BEZOARS IN GASTRO-INTESTINAL TRACT WITH A CASE REPORT

BY

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The trichobezoar or Hair-ball of the stomach in animals has been known since centuries. It was known to Hindus as early as twelfth century B.C. and was used by them as a superstition as one of the most precious medicines for treatment of many diseases, *e.g.*, for neutralising poisons, rejuvenating old age and for the prevention of many afflictions. Probably the word bezoar is derived from Arabic word 'Badzehr' or Persian '*Padzehr*' which means antidote or counterpoison. Their precious value can be realised from the fact that they were preserved in frames of gold and diamonds as amulets. They probably escaped medical recognition for long when trichobezoar in man was authentically recorded first by Baudamant in 1779. Since then much work has been done and so far 187 cases (including this case) of trichobezoar have been authentically collected. The first case of phytobezoar was recorded by Quain in 1854. Schonborn in 1883 removed trichobezoar for the first time surgically. Thornburn in 1886, for the first time made a correct pre-operative diagnosis of hair-ball. The first published description of roentgenologic appearance of a bezoar was made by Holland in 1913. Most work has been done by De-Bakey and Oschner who by 1939 collected 311 cases of all types of bezoars from the world record.

Classification

Following is the classification of bezoars according to the nature of the material which forms the major part of its contents.

(1) *Trichobezoars :* Here hair forms the predominating part. The hair may be derived from cow, goat, horse, human hair or bristles, string and wool-threads.

The hair forming these bezoars are of varying length which intricately interlace with one another so as to form a delicate lattice cemented by gastric juice and moulded on the whole of stomach forming a 'Hair cast'. Their weight varies, the average being about 1.6 lbs. The heaviest bezoar recorded by Davies (1921) weighed 6½ lbs. Usually single, they may be multiple. Their surface is covered by a thick mucoid substance, and their meshes may be filled with undigested food particle of various kinds especially fat (which is not digested by gastric juice). They are offensive smelling because of fermentation, putrefaction and decomposition of the food particles they contain. Their colour varies from dark-green to black, irrespective of the character of swallowed hair, due to the colour changes produced by the action of putrefying gases on hair. Extremely rarely they are reported to have extended beyond stomach to oesophagus and duodenum. However their extension beyond duodenum has probably not been reported except in a case of Malhotra and Manchanda in which also a daughter bezoar had got separated from the parent one and was connected to it by a thin streak. In our case, the single bezoar was filling the whole of stomach, duodenum and proximal one foot of jejunum in a single mass.

They are commonest of all bezoars and formed 172 of DeBakey and Oschners collections. The number of total cases reported till August 1957 was 188 and this probably is the 189th case on world record.

(2) *Phytobezoars :* They are formed by substances of vegetable origin, *e.g.*, stems, roots, large variety of foods, coconut fibres and cotton-threads (in cotton-workers). The commonest amongst them are those of persimmon which contain skin, pulp and seeds of wild persimmon (ingested by hunters, golfers etc. who are engaged in outdoor activities).

They are smaller than hair-balls, and vary in shape considerably *e.g.*, spherical, cylindrical, conical, oval, pear or brick shaped etc. and form a partial cast of stomach. Their weight varies from 1 Gm. to 200 Gms. and colour from dark black, brown or greenish yellow on outer surface to a lighter one on inner surface. They are hard but friable and their smell varies from putrid or sour to aroma of fermenting fruits.

They form second commonest series amongst bezoars and were 126 of DeBakey and Oschner's collection.

(3) *Trichophytobezoars :* These contain materials derived both from animal and vegetabl origin.

(4) *Concretions :* They are formed by substances which are not of biological origin. Examples are concretions of bismuth carb. magnesium, and sodium carbonate, paraffin, salol and shellac, paraffin being the commonest. They are found in stomach of persons who deal with the above substances. They are very rare and amongst them, paraffin concretions are more predominant. They formed 13 of DeBakey and Oschner's collections.

Aetiology

Age Incidence : Though hair-balls have been recorded at as young an age as 1 year and as old as 65 years, the commonest age-group in which they are found is 15 to 30 years. About 80% of them occur between 15 to 30 years. Persimmon balls however are more common in middle age.

Sex Incidence : Almost 90% of the cases are women, probably because hair in girls are longer than in males, and thus they (girls) predispose themselves to chewing and swallowing hair. However Maes (1928) did not agree to this explanation because he did not find decrease in their incidence after the ladies adopted the custom of keeping their hair bobbed.

Mental activity : About 14% of these children suffer from some sort of psychic and mental disturbances, while in 50% of cases history of habit of trichophagia (swallowing hair) can be obtained. Golden Lewis considers this habit as a symptom of personality disorder, *e.g.*, nail-biting, thumb-sucking etc.

Mechanism : These hairs are swallowed in small amounts for a prolonged period. Probably the stomach is unable to expel these hairs, so that these wisps go on accumulating in rugaes and folds of the stomach and gradually form into a ball.

The mechanism of formation of persimmon bezoars has been largely studied by Izumi, Isida and Iwamato, who provide the following reasonable explanation. According to them, ripe persimmons contain a large amount of insoluble 'shibuol' and a little amount of soluble 'shibuol' an element which is precipitated by gastric juice and other mineral acids, into a sticky mass. Ghont has independently confirmed the necessity of hydrochloric acid in the production of bezoars. The formation of these persimmons bezoars in contrast to trichobezoars is not gradual but immediate, resulting from eating large amounts of this fruit at a time especially on an empty stomach.

Concretions are formed by chemical process. Shellac is soluble in alcohol but insoluble in water, hydrochloric acid and alkaline solutions ; hence it is easily precipitated when alcohol is diluted and absorbed in the stomach.

Clinical features

The trichobezoars give rise to vague dyspeptic symptoms due to gastric irritation generally only after they have formed a tremendous mass. The character and intensity of these symptoms depends upon size, nature and age of the bezoar and presence of complications. These symptoms are in the form of anorexia, weakness, loss of weight, abdominal discomfort and later on, nausea and vomiting and the patient may complain of paroxysmal attacks of epigastric pain. At this stage constipation may alternate with diarrhoea. In 6% of cases there is history of haematemesis due to presence of gastric ulcer.

In 90% of cases the bezoar can be palpated as a mobile, well defined, uniform, J shaped and smooth intra-abdominal swelling extending from one hypochondrium to the other. Occasionally its parts may be passed in stools.

If hair ball is near pylorus, a condition resembling pyloric stenosis may result with paroxysmal vomiting and epigastric pain.

On the other hand, persimmon bezoars start with symptoms of acute gastro-intestinal upsets and being small are not palpable.

Occasionally a small bezoar may break from the parent one and may pass in bowels giving rise to intestinal obstruction. Final picture presented may be due to ulceration of stomach, perforation or peritonitis. Occasionally the patient may die of inanition due to the size of the '*tumour*.'

In general, symptoms and signs of bezoars are not easily distinguishable from those of other abdominal conditions and hence a preoperative clinical diagnosis is not made.

Investigations

The following investigations should be made :—

(1) Blood : Often shows mild degree of hypochromic anaemia and mild leucocytosis.

(2) Gastric test meal : In trichobezoar it is generally normal or of low gastric acidity and sometimes may show presence of hair. In phytobezoar, the values of hydrochloric acid are often high and thus they corroborate with the mode of their development.

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Fig. 3
X-Ray taken 2 hours after meal.

Fig. 2
X-Ray taken 1½ hours after meal.

Fig. 1
Barium meal, X-Ray taken immediately.

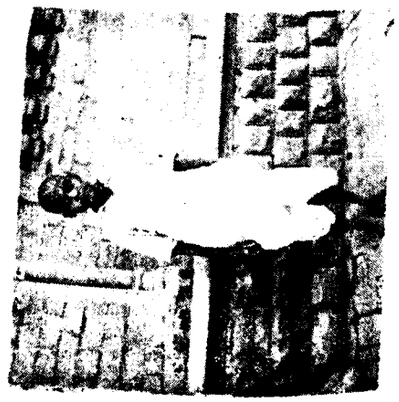

Fig. 5
Photo after the Operation.

Fig. 4
Hairball in the stomach, duodenum and small intestines.

(3) Stool examination : Occasionally reveals strands of hair in trichobezoar.

(4) Gastroscopy : May sometimes be helpful.

(5) *Radiology :* is the most important procedure employed in the diagnosis of bezoars.

(*a*) Plain X-Ray abdomen : may occasionally show an area of increased density conforming to the shape of stomach which suggests the possibility of trichobezoar.

(*b*) Barium meal examination : is the most important and useful of all the procedures employed for their diagnosis. *Holland* has described the fluoroscopic and roentgenographic appearance with barium meal as mentioned below :

" Typically, fluoroscopic examination, performed in the patient in erect position, shows the swallowed barium held up in the cardiac end of the stomach for a few seconds as though forming a cap on something inside the organ, then suddenly diffusing slowly downwards on either side of the non-opaque foreign body following the regular contours of the greater and lesser curvatures to map out the normal contour of the stomach. At the end of 6 hours most of the barium had left the stomach but usually a sufficient amount remains attached to the mass to outline the stomach in a lace-like pattern."

Greater and especially lesser curvature must always be carefully examined for niche of the associated ulcer. If the ball extends to duodenum, the fine reticular pattern may extend to that part of bowels. In persimmon balls, the mottling is associated with local coarser densities and as they are smaller, they can be shifted from one position to the other in stomach by simple manipulation.

Complications

The complications and their incidence in trichobezoar as based on 188 collected cases of Malhotra and Manchanda have been described as follows :

1. *Intestinal Obstruction :* (*a*) By parent hair ball—1 case. This was described by Trafford in which the trichobezoar left the stomach and became impacted in ileum causing intestinal obstruction which resembled intussusception.

(*b*) By daughter hair ball—1 case. This was described by Malhotra and Manchanda where a part got separated from the parent one, being connected to it by a small strand, and caused intestinal obstruction in ileum.

(*c*) By parent and daughter hair-balls—1 case.

2. *Gastro-duodenal ulcerations :* 15 cases. In 80% of cases they are found on lesser curvature near the angle. According to Oschner they are found in 9.6% cases of trichobezoar.

3. *Perforation of ulcer :* 5 cases. They form 2.9% of all cases and 33% of cases having ulcer.

4. *Peritonitis :* 6 cases.

5. *Inanition :* 13 cases.

Intestinal obstruction is however commoner in phytobezoars because they are smaller and can pass comparatively easily from stomach to small intestines.

Diagnosis : The diagnosis can be made only when the condition is suspected, by clinical symptoms *i.e.*, presence of a freely mobile intra-abdominal lump and the symptoms of dyspepsia in a young girl in whom history of trichophagia can be obtained. Persimmon bezoars are however common in middle aged people and often produce symptoms of acute gastro-enteritis. Barium meal examination will confirm the diagnosis.

The conditions with which it can be confused are as follows :

1. Left upper intra-abdominal lump may be suspected to be a renal tumour, omental or mesenteric tumour or cancer of stomach.

2. Lump + anaemia + slight leucocytosis may make the clinician to suspect lymphosarcoma, leukaemia or Banti's disease.

3. Symptoms of dyspepsia, especially in persimmon bezoars may be confused which chronic cholecystitis, peptic ulcer or carcinoma of stomach.

4. Roentgenological examination which shows such diffuse reticular outline may be mistaken for diffuse gastric polyposis.

Treatment : Once the diagnosis has been made, the bezoar must be removed by gastrotomy. Fatality rate, recorded to be 10.4% by DeBakey and Oschner, seems to be decreasing with the advent and use of antibiotics.

Peptic ulcer, if in association heals rapidly once the bezoar has been removed. Treatment of other complications is on the same lines as the treatment due to other causes.

Case Report

R.A. a young Hindu male child of 11 years was admitted in S.N. Hospital, Agra on 1st October 1957.

Complaints :
Patient complained of lump in abdomen for 1 year.

Past History :
There was history of dysentery 1 year back.

Family and Social History :
There was no history of Tuberculosis in the family. Patient was vegetarian and did not give history of having swallowed any abnormal thing *e.g.* threads or hairs.

Present History :
Patient stated that he got loose motions containing blood and mucous 1 year back. This lasted for 15 days, after which he noticed a small swelling in upper part of abdomen. One month after the appearance of this lump, he started with attacks of severe colicky pain in abdomen, most marked in the upper part each attack lasting for 2 hours and not accompanied by vomiting and having no relation with the food, or motions. They used to be relieved by themselves. The attacks used to occur at an interval of 2 to 3 months and he had 5 such attacks. The lump had been gradually increasing in size. He further said that every morning after a motion he used to get slight pain in abdomen in the left hypochondrium which was relieved by taking some food.

General Examinations :
Young boy of weak build and very intelligent though not educated.
Slight Anaemia.
No Jaundice.
No glandular enlargement.
Pulse—80/min.
Temp. 98.4 F.
B.P. 104/70.

Local Examination of Abdomen :
Site—A hard Intra-abdominal Lump in epigastrium and going to left hypochondrium, deep to the subcostal margin.
Shape—Cone shaped with apex towards right side.
Surface—Smooth except a ridge in the middle.
Margin—Well defined.
Consistency—Stony—hard.
Mobility—Mobile in up and down direction and from side to side.
The lump did not move with respiration.
It was not continuous with liver or spleen.
Percussion note—Resonant in front of the lump.

Systemic Examination :
Heart—Normal.
Lungs—Normal.

Investigations :
Stool—No abnormality seen.
Urine—Showed calcium oxalate crystate.
Blood—R.B.C.—3.35 millions/cmm.
Hb. 10 Gm.%
Total W.B.C.—12400/cmm.
Diff. W.B.C. Poly. 60%. L. 29, E. 10%
Plain X-Ray abdomen—No abnormality seen.
Barium enema—There was a filling defect in the ascending colon, near the hepatic flexure.

Barium meal :
The meal passes normally through the oesophagus. It fills the stomach slowly and irregularly and certain masses in the lumen of the stomach are seen protruding into the barium shadow causing multiple filling defects. The 1½ hours skiagram shows the meal in the jejunum and the ileum, but here also the lumen is not completely filled by the meal due to multiple intra-cavitary projections. Radiologically the condition is suggestive of multiple polyposis—although the skiagrams are not typical of the same.

Diagnosis : No tentative diagnosis of the intra-abdominal lump was arrived at. Laparotomy was done on 21st October 1957, and the abdomen was opened under general anaesthesia through the left upper paramedian incision. The stomach presented itself. The lump was felt inside the stomach which was opened through a 3" long incision on its anterior wall. The lump was found to be a mass of hair which was removed. It was having the shape of the stomach and had a tail containing hairs, and threads about 3 feet long, filling the whole of duodenum and upper part of jejunum. The stomach was closed in three layers. Ryles tube was passed in stomach and the abdomen was closed.

Progress

Patient was kept on Crys. Penicillin 1 lac. 4 hourly and streptomycin 1 gm. I.M. for 3 days. Then Dicrysticin 1 Gm. was given intramuscularly for 2 days. Ryle's tube was removed on the third day.

The patient started drinking milk on the third day and taking semi-solids on the fourth or fifth day.

He was discharged cured after the abdominal stitches had been removed on the ninth day. He had a good appetite and he gradually took solid food and gained in weight.

Summary

The literature of bezoars in the gastro-intestinal tract has been reviewed.

A case of trichophytobezoar has been reported who had the following special features :

(*a*) He was a very intelligent male child, without any history of foreign material having been swallowed.

(*b*) The bezoar was filling not only the stomach but also the whole of duodenum and upper one foot of jejunum.

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MALIGNANT LYMPHOMAS OF THE STOMACH

(A Report of four cases)

BY

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Sarcoma of the stomach is not a common gastric neoplasm, but survey of the literature conveys the impression that it occurs fairly frequently and accounts for about 2% of all gastric malignancies. Higher incidence of 3.5% has been reported by Marshall and Meissner[2] in their series. Yarnis and Colp[6] found an incidence of 3.2% in a series of 250 cases of gastric neoplasms. Snoddy[3] reviewed the literature in 1952 and collected 474 cases of sarcoma of stomach and added 34 of his own.

These tumours though rare are being reported with increasing frequency and are of interest because of the diagnostic difficulties they offer, and better prognosis if treated early.

Sarcomas occur in a younger age group, not infrequently present as palpable tumours and, because of their size and the extent of stomach involvement, may appear as inoperable carcinomas. Majority of these tumours are, however, not diagnosed before operation. But it is important to consider them in the differential diagnosis of gastric neoplasms, recognise them at operation and treat them accordingly because surgical treatment offers a much better prognosis in sarcomas than in carcinomas.

Pathology

Sarcoma of the stomach usually arises from smooth muscle or the lymphoid tissue. The malignant lymphoid tumours may be divided according to Warren and Lulenski[5] as follows :

(i) Hodgkin's Disease.
(ii) Reticulum Cell Sarcoma.
(iii) Lumphosarcoma.
(iv) Malignant Lymphoma.

It is interesting to note that four cases of sarcoma of stomach were met with in the S.M.S. Hospital in two years, (1956–57), all of them being of lymphoid origin. Malignant Lymphoma including Hodgkin's disease, Reticulum Cell Sarcoma, Giant follicular lymphoma and lymphocytic leukemias are generalised systemic diseases, and involvement of the stomach or Gastro Intestinal tract may take place secondarily. But in the cases reported below, there was no evidence of generalised disease being present.

Case Report

Case No. 1.
Mr. B.R. a 67 year old H.M. admitted to S.M.S. Hospital, on 21st January 1956 complaining of epigastric pain (pricking) for 6 months. Pain intermittent in character and was relieved on taking meals. But one month prior to admission it had become constant. No vomiting, no nausea, no haematemesis.

On examination—a man of 67 years, well built and fairly nourished. No jaundice. Tongue moist and slightly furred. Teeth dirty. No lymphnode enlargement. Pulse 80/mt. Resp. 20/mt. B.P. 125/80 mm. Hg.

Physical examination of the abdomen did not reveal any palpable lump in the epigastric region. Tenderness in the epigastrium present. Liver and spleen not palpable. No other relevant findings.

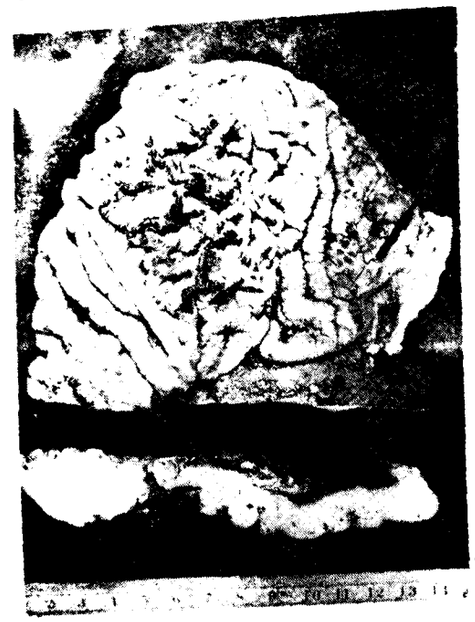

Fig. 1
Diffuse infiltration of the stomach wall in lymphosarcoma shown below. Arrow points to the niche.

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Investigations

Blood picture	...	R.B.C. 3 million/cu.mm. Hb. 12 G% T.L.C. 19000/cu.mm., P 75%, L. 25%.
Stool	...	Positive of occult blood.
F.T.M.	...	*High total acidity.* Blood and Mucus present in all specimens.
Barium Meal	...	Filling defect in the stomach.

Exploration of the abdomen revealed a growth in connection with the anterior wall of the stomach. Subpyloric group of glands enlarged. No other metastasis. A subtotal gastrectomy was performed.

Pathology Report

Distal half of the stomach showed one ulcer 2 cm. in diameter with indurated margins, situated in the centre of the specimen. The wall of the stomach was diffusely thickened with prominent rugae. It was firm in consistency and on cutting, a localized whitish area was seen beneath the mucosa (Fig. 1). Microscopically diffuse masses of spheroidal cells almost of uniform size are seen beneath the mucosa. The mucosa was lifted and also infiltrated by these round cells. Musculature is atrophied and infiltrated by the same type of cells which have also involved the serosa. The type of cell forming this neoplasm is a round cell, a little larger than a lymphocyte, with large vesicular nucleus and prominent nucleoli. The lymphnodes removed from the specimen show evidence of Giant follicles proliferating to the extent of fusing with each other and at places forming a diffuse mass of spheroidal cells as seen in the stomach. *Diagnosis :* Lymphoblastic lympho-sarcoma primarily of the stomach with involvement of the regional lymphnodes.

Case No. 2 : M.B. 40 year old female admitted to S.M.S. Hospital on 1st September 1956 complaining of a painful lump in the epigastrium of four months duration, loss of appetite and generalised weakness for the same period. The first symptom to appear was epigastric pain which was followed by a lump gradually increasing in size and which has attained the present dimensions. No hematemesis and melaena. On examination an adult female poorly built and nourished ,lightly anaemic. No lymphnode enlargement. Pulse 96/mt., Resp. 12/mt., B.P. 100/60 mm. Hg.

Physical examination revealed a large intra-abdominal swelling 10 cms × 7.5 cms. in the epigastric region, moving on respiration, tender, firm with well defined margins and smooth surfaces. Liver and spleen not palpable. Other systemic examination did not reveal any abnormality. A clinical diagnosis of carcinoma of the stomach was made.

Investigations : R.B.C. 3.5 million/cu.mm., Hb. 9.5 G%, T.L.C. 8000/cu.mm., P 70%, L 25%, M 3%, E 2%. Stool was negative for occult blood. F.T.M. showed hypochlorhydria. Barium swallow showed filling defect at greater curvature near the pylorus.

Exploration of the abdomen, done on the 24th September 1956, revealed an inoperable tumour of the stomach involving the pyloric part, adherent to the pancreas and metastatic lymphnodes present all along the lesser and greater curvature of the stomach. A biopsy of the tumour was taken and an antecolic long loop Isoperistaltic Gastro-Jejunostomy was performed to relieve the obstructive symptoms.

Pathology Report : The cut surface of the lymphnode shows complete loss of architecture and marked proliferation of reticulum cells, which also infiltrate the capsule. The cells have pale staining cytoplasm and vesicular nuclei. Diagnosis Reticulum cell sarcoma of the lymphnode primarily that of stomach.

Case No. 3 : Mrs. A.B., 50 year old house-wife, admitted on 8th November 1956 complaining of epigastric pain of three months duration. The pain radiated towards the right hypochondrium and spread all over the upper abdomen accompanied by nausea, and was relieved by vomiting. Fifteen days prior to admission the pain had become constant and dull aching in character. Patient had some relief with milk and her diet now consisted mostly of milk. She had no hamatemesis. On examination the patient was fairly built and nourished. Conjunctivae pale. Tongue moist and coated. No lymphnode enlargement. Pulse 88/mt., Resp. 20/mt., B.P. 120/90 mm. Hg.

Physical examination : Revealed a firm ill-defined lump about 7.5 cms × 6 cms. in the epigastrium more to the right side of the midline. Tenderness present. Lump moves on respiration. Liver and spleen not palpable. No other relevant findings. Clinical diagnosis, Cancer of the stomach was made. Laboratory investigations were non-contributory. F.T.M. showed hypochlohydria. Filling defect in the pyloric region was seen in the barium meal examination.

The patient was operated on, on 28th November 1956 and a large tumour involving the pyloric part of the stomach and adherent to the pancreas was found. Two enlarged lymphnodes were also present along the lesser curvature. No metastasis in the liver, hence a sub-total gastrectomy was performed.

Pathology Report : A large infiltrative type of growth near the distal end of the stomach (Fig. 2). Rest of the stomach was also thickened but not so much thick and hard as the growth itself. Mucosal surface of the growth was necrotic and showed several areas of haemorrhage. The wall was 2 cm. thick and whitish in colour. Microscopically a diffuse infiltration by round and Pleomorphic cells seen mainly in the submucous region. The mucosa is lifted, atrophied and infiltrated by these cells. Infiltration of the muscularis is also evident, at many places involving the serosa also. Diagnosis-Reticulum cell sarcoma of the stomach. (Fig. 4).

Case No. 4 : M.D., 65 year, H.M., admitted on 4th July 1957 complaining of pain in the epigastric region for the last 3 months, noticed lump in the epigastrium 1½ months prior to admission and vomiting for the last 2 months. On examination a man of 65 years moderately built and fairly nourished. Pulse 80/mt., B.P. 100/70 mm. Hg. No glandular enlargement. Physical examination of the abdomen revealed a firm, ill-defined lump 6 cms. – 5 cms. in the epigastrium, moving with respiration and non-pulsatile. Liver and spleen not palpable. Clinical diagnosis of Cancer of the stomach was made. Laboratory investigations were non-contributory except that there was absence of free acidity in the F.T.M. examination and filling defect was observed in the barium meal examination.

Exploration revealed a huge mass in relation with the stomach involving the lesser curvature of about half way to the cardiac end. Tumour was adherent to the transverse colon and there was a gastro-colic fistula. Biopsy was taken from the tumour.

Pathology Report : Diffuse mass of small round cells, resembling lymphocytes extensively infiltrating the connective tissue. There is monotony of cells and no attempt of orientation to any type. Diagnosis—Lymphosarcoma of the stomach. (Figs. 3 and 5).

Fig. 2
Diffuse infiltration of stomach by reticulum cell sarcoma with superficial ulceration (arrow) and haemorrhagic spots on the mucosa.

Fig. 3
Lymphosarcoma stomach. Diffuse sheets of lymphocytes infiltrating the wall of the stomach and pushing mucosa on the top. X 100.H.E.

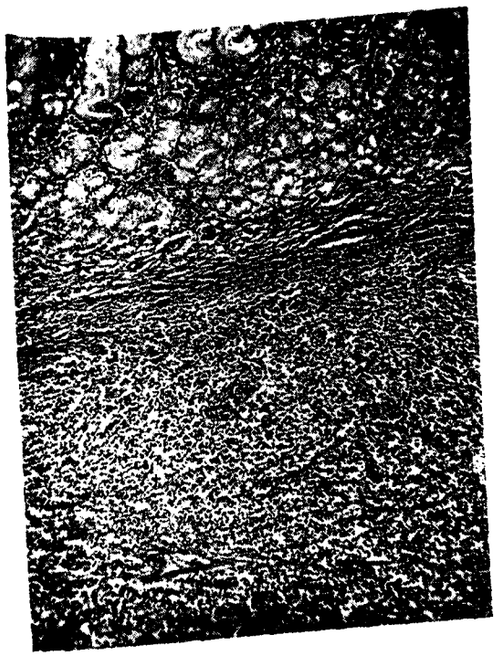

Fig. 4
Reticulum cell sarcoma stomach seen as diffuse sheets of reticulum cells beneath the mucosa. X 100.H.E.

Fig. 5
Lymphosarcoma of the stomach showing sheets of lymphocytes infiltrating diffusely. × 270. H. & E.

Comments

Although sarcoma of the stomach is said to occur more commonly in younger group of patients Farmer and Hertzog[1] reported 7 cases of primary lymphosarcoma of the stomach and found an average age of 56 years. Marshall and Meissner[2] in a review of 41 cases of sarcoma of the stomach found 43 years as the average age. The average age of patients in this series was 55 years, the youngest being 40 years and the oldest 63 years. Sex distribution was equal in number. The sex ratio from the literature averages from 1.5 to 2 males to 1 female, while in 7 cases reported by Farmer et al, ratio was 6 males to 1 female.

The presenting symptoms were epigastric pain, gradually increasing lump and anorexia. Epigastric pain is a prominent early symptom of sarcoma of the stomach. Pain was relieved by vomiting in two of our cases and one was relieved after taking meals. Lump was palpable in 3 cases on admission. Melaena was present in 3 cases while hematemesis was absent in all. Hypochlorhydria was present in 3 cases while total acidity was increased in one case.

Roentgenographic studies were only helpful to the extent that they revealed filling defect on barium meal examination and all of them were diagnosed as carcinoma of the stomach. 3 of our cases showed filling defect and in one case doubtful evidence of an ulcer was present in the pyloric region. (Case No. 3).

Involvement of the pyloric part and lesser curvature was predominantly observed in 3 cases, and regional lymphnodes were enlarged in all the 4 cases but distant metastases were not observed in any of the cases. Histologically two cases were Reticulum cell sarcoma, one lymphoblastic lymphosarcoma and one lymphocytic lymphosarcoma.

The treatment of choice of gastric lymphosarcoma is radical surgery followed by postoperative irradiation. "Enlargement of the regional lymphnodes should not be a contraindication to radical surgery as these nodes usually prove to be reactive hyperplasia rather than matastatic involvement." (Hertzog et al).

Follow-up studies were not available in any of the cases. Though Marshall and Meissner[2] report a five year post-operative survival rate of 33% following total gastrectomies and 42% following partial gastrectomy for lymphoid tumours of the stomach excluding Hodgkin's disease. Thorbjarnarson, Beal and Pearce[4] in a series of 32 cases with primary malignant gastric lymphomas reported 53%, 5 years survival rate. Recently in 7 cases reported by Farmer et al only one patient survived more than 3 years following surgery.

Summary

4 cases of primary malignant lymphomas of the stomach have been reported. A short review of the literature is given. Age incidence and clinical features differ little from carcinoma. Presenting symptoms in all cases were epigastric pain and lump in three of the cases. Diagnosis of the sarcoma could not be made pre-operatively. Roentgenographic examination was found to be of little help in the diagnosis.

Even seemingly inoperable cases should be explored, as sometimes sarcomas present themselves as large palpable tumours, and one is gratified to find the lesion resectable on opening the abdomen. The treatment of choice is radical surgery followed by Deep X-ray. Prognosis is considered to be better than for gastric carcinomas.

ACKNOWLEDGEMENTS

We wish to express our thanks to Professor G. L. Talwar, F.R.C.S., for permitting us to publish his case, (Case No. 1) and also our thanks are due to the Superintendent, Dr. L. R. Sarin, M.R.C.P., S.M.S. Hospital, Jaipur for permitting us to publish these cases from the hospital records.

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PROLAPSE OF THE GASTRIC MUCOSA

(A Case Report)

BY

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Prolapse of the gastric mucosa implies the protrusion of the redundant and loose gastric mucous membrane through the pylorus into the duodenum. Schmieden[21] was the first to describe this entity. Eliason and Wright[6] reported the second case. The purpose of this report is to review briefly the literature on this condition and to report a case.

Incidence of prolapse of gastric mucosa has varied widely in cases subjected to radiological examination of upper gastro-intestinal tract. Scott[20] reported it to be 1%; Manning and Gunter[12] 1.24%; Hawley[8] 2.8%; Cove and Cruphey[1] 3.3%; Wellens and Spyckerelle[22] 5%; Appleby[1] 7.0%; Ferguson[7] 7.7%; and Rappaport[18] 15.5%. It may occur at any age but the maximum incidence is in the 4th and the 5th decade, occurring predominantly in males.

Aetiology

Aetiological factors responsible for its production have not yet been fully understood. Eliason and Wright[6] explained its development by the occurrence of a low grade mucosal inflammation in the pyloric antrum which produced local hypertrophy of the mucous membrane. The mucosal folds get elongated by the action of gastric peristalsis and pressure of food contents, and continue to do so till they prolapse through the pylorus into the duodenum. To further reinforce the above hypothesis Bohrer and Copleman[3] and Norgore and Shuler[14] found evidence of gastritis in surgically treated cases of prolapse.

Rees[19] believed that pyloric narrowing preceded the mucosal change. The gastric hyperperistalsis necessary to force contents through the narrow pylorus leads to loosening of the attachment of mucosa to muscularis propria. This redundant mucosa when subjected to repeated trauma leads to chronic inflammation and hypertrophy which is the precursor of prolapse. Scott[20] and Cove and Curphey offer a mechanical basis based on hypermobility of gastric mucosa. This hypermobility may be congenital or may be produced by abnormal gastric motility which, over a prolonged period, loosens the mucous membrane from submucosa to such an extent that now gastric mucous membrane can prolapse into duodenum. Scott believed that this abnormal gastric peristalsis could be initiated by neurogenic and/or chemical stimuli. According to Wellens and Spyckerelle[22], in systemic disorders producing oedema, the oedematous gastric mucous membrane is more liable to prolapse and this may explain the occurrence of gastro-intestinal symptoms in such disorders.

The clinical picture is non-specific and the severity of symptoms depends on the degree of prolapse and state of prolapsed mucosa. Epigastric discomfort or severe pain in the epigastrium or right hypochondrium, coming on at varying intervals after food, is the most common symptom. The pain may radiate to the back. There may be accompanying abdominal distension, nausea and vomiting. The pain may be relieved by food or occasionally by alkalies. Severe degree of prolapse may lead to temporary complete pyloric obstruction with attacks of nausea and vomiting (Eliason, Pendergrass and Wright[5]; Meyer and Singer[13] Rees[19].) Slow oozing of blood may occur from ulceration of prolapsed mucosa but severe bleeding is rare (Archer and Cooper[2]).

The diagnosis is generally based on radiological examination. The suggestive finding is the presence of a lobulated filling defect in the base of bulb of duodenum. The gastric rugae which are thickened and smooth are seen passing from the antrum transpylorically into the bulb.

Conservative therapy constituting a bland diet, alkalies and antispasmodics may relieve symptoms in early cases. However, surgical therapy is the only choice in late cases. Continued bleeding and evidence of pyloric obstruction are absolute indications for surgical intervention (Mackenzie[11]) Commonly employed surgical procedure is resection of prolapsed and redundant mucosal folds with pyloroplasty. Alternative procedure is partial gastrectomy which is especially useful in cases

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with bleeding (Kohler[9]). The results of surgical therapy appear to be satisfactory.

Case Report

L.J., male, aged 17 years, complained of frequent attacks of moderately severe right hypochondriac pain, radiating to right lumbar region for the last 7 years. The pain used to last for a few hours and was accompanied by vomiting. Investigations in November, 1953 : urinalysis revealed no abnormality ; Haemoglobin was 9.5 Gms.% ; Total leucocyte count 6,500 per cmm., polymorphs 55% ; Lymphocytes 41% ; Monocytes 1% ; Eosinophils 3% ; Serum amylase 320 somogyi units per 100 ccs. ; Urinary diastase 60 Wohlgemuth units per cc. ; Cholecystography revealed a normally functioning gall-bladder ; barium meal study of gastro-intestinal tract did not reveal any abnormality. In January 1954, endocholedochal sphincterotomy with cholecystectomy was performed elsewhere. In August 1954 he again started getting similar attacks of pain, urinalysis then revealed no abnormality, serum amylase was 133 somogyi units per 100 cc. ; Urinary diastase was 20 Wohlgemuth units per cc. Intravenous cholangiography with biligrafin revealed normal biliary ducts, barium meal study of upper gastro-intestinal tract showed no evidence of disease. Since then he was getting frequent attacks of severe colicky epigastric pain which was temporarily relieved after taking milk or tea. There was occasional accompanying vomiting which at times relieved pain. During this period he had haemetemesis twice which was moderate. His appetite was lost and he had lost considerable weight. On 13th August 1956 he was admitted to our wards. While under observation in the ward his symptoms became severe and persistent. He was in constant agony tossing about in bed, only relieved temporarily by vomiting or by analgesics. During this period he again had mild haematemesis a third time. Following investigations were done.

Urinalysis revealed no abnormality ; nothing abnormal was detected after repeated examination of stools ; serum amylase was 200 somogyi units per 100 cc. ; urinary diastase was 20 Wohlgemuth units ; Gastric analysis showed a high free and total acidity ; Fasting gastric contents estimated daily over a period of 8 days, varied from 90 to 150 ccs. Intravenous cholangiography with biligrafin normally outlined the biliary passages. On barium meal study an ulcer niche was seen in the first part of duodenum with evidence of gastric stasis, considerable meal having been present in the stomach 10 hours later. Barium meal study again on 16th September 1956 showed suspicion of duodenal ulcer with gastric stasis. There was also a suggestion of prolapse of gastric mucosa into duodenum.

At operation on 18th January 1957 many adhesions were found in the region of pyloric end of stomach and duodenum as a result of previous operation of cholecystectomy and sphincterotomy. No evidence of peptic ulcer or any other disease was detected in stomach or duodenum. Partial gastrectomy by Polya Hofmeister technique was done. The resected specimen was examined, and on looking from the pyloric end it appeared that two folds of mucous membrane were protruding into it. The stomach was then slit leaving pylorus intact and it became clear that there were loose folds of mucous membrane which could be prolapsed into duodenum (Figs. 1 and 2). These folds could be easily lifted up with forceps. In the rest of the pyloric antrum the mucosa was not loose and no folds were visible.

He reported for follow-up on 5th January 1958. There is complete relief from symptoms, his appetite has returned to normal and he has gained weight. He has gone back to his school and is now studying in ninth class.

Fig. 1

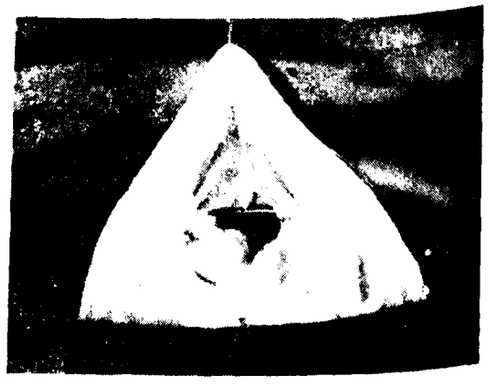

Fig. 2

Figs. 1 & 2 : Resected specimens showing fax and redundant prepyloric gastric mucosa tending to prolapse into duodenum.

Comments

The patient under discussion is an adolescent male aged 17 years who started getting symptoms at the age of 10 years and suffered for 7 years. The diagnosis presented the main difficulty in view of the non-specific nature of symptoms. He was misdiagnosed twice, first as a case of pancreatitis and later as peptic ulcer. Repeated barium meal studies for first 5 years failed to reveal any abnormality but in the last series of examinations diagnosis

of peptic ulcer with suspicion or prolapse of gastric mucosa was reported. These diagnostic mishaps are not unknown in other reported series. Scott[20] misdiagnosed the condition in every case at initial barium meal examination and reported duodenal ulcer in 11 of the cases, duodenitis in two and gastric ulcer in one case. Lichstein[10] explained these discrepancies in radiological diagnosis by emphasizing that the condition is a result of dynamic state that subsides and recurs with peristaltic activity and therefore the appearance of the defect is changeable from time to time and may even disappear entirely. Despite these pitfalls radiological diagnosis remains the only confirmatory evidence. Operative findings did not reveal any evidence of active or healed peptic ulcer, but on opening the stomach after resection antral mucosa was found to be loose and redundant, could be picked up with forceps and prolapsed into duodenum. Examination of the resected specimen supported the diagnosis. The unusual features in the case history were repeated attacks of haematemesis and radiological evidence of gastric stasis without accompanying dilatation of stomach to prove established pyloric obstruction. Conservative measures did not provide him with lasting relief ; instead the symptoms were progressive. Partial gastrectomy has relieved him completely.

Summary

A case of prolapse of gastric mucosa has been reported. The subject has been briefly reviewed with a view to stress certain features of clinical importance.

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ORTHOPAEDIC SECTION

ROLE OF DIETARY PROTEINS IN THE HEALING OF EXPERIMENTAL FRACTURES

BY

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Two factors are involved in the healing of fractures (*a*) Biological and (*b*) Mechanical. Whereas in the Orthopaedic literature plenty of attention has been paid to the elucidation of the mechanical factor, very little is known about the biological factor. According to Sir Harry Platt[1]—still have very little information regarding chemical processes, which have any obvious application to treatment—Processes of ossification is a chemical change taking place in osteoid tissue, which is thus caused to harden and to become bone..... 'In a broad manner, we can say that there are two major processes, laying down of the organic matrix and subsequent mineralisation.[2] According to Pheimister[3] the intermedullary callus is usually transferred into bone by fibrous or inter-membranous ossification with the formation of little or no cartilage, while transformation of the peripheral callus is partly by inter-membranous and partly by endochondral ossification. The intermediary callus, being most exposed to the trauma of motion and compression between fragment ends, may undergo more or less necrosis and transformation into fibro-cartilage and is the last part of the callus to ossify. The laying down of the protein matrix is the first part in bridging the gap and for the formation of osteoid tissue. Whether the quality of protein has anything to do with the regeneration of osteoid tissue or not cannot be answered either on theoretical considerations or such indirect observations as made by Pandit[4] during fluorises enquiry conducted in a community of low nutritional status. We are now in possession of exact figures relating to the amino acid composition of collagen, gelatin and osseo-mucoid[5] and the availability of such amino acids in dietary materials.[6] It is doubtful whether a correlation between the two may be of any help. In the case of trypsinogenesis in cases of Kwashiorkor[7,8]. it has been shown that it is not the administration of the required amino acids but some building blocks or proteins of particular nature that bring about regeneration of pancreatic enzymes. For example casein has been found to be better than any other type of protein. It was therefore thought worthwhile to see the effect of various cereal proteins in the regeneration of the organic matrix. Moreover, patients coming to our hospitals depend upon low quality cereals for the supply of their proteins. How much does it influence healing of fractures that are set and plastered in the outdoor section of the Orthopaedic department, is a question whose answer may be available after evaluation of the results obtained in the present series of experiments.

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Experimental procedure

Experiments were performed on rats. These were divided into three batches. One batch was kept on a balanced diet with milk as the chief source of protein. The other batch was kept on grams and a third on bajra, along with minerals, vitamins and other nutrients to make the diet uniform and well balanced in all respects except for the cereal proteins which differed from batch to batch while the quantity of proteins administered remained the same.

Fractures were produced in the following manner. A wooden platform had two brass plates so protruded that the thigh of the rat could be easily placed in between the two plates and parallel to them while the animal lay resting on the platform. The thigh was kept in position over two blunt bevelled edges and could be clamped. A hook was placed at the ventral aspect of the thigh and carried at its other end a pan for loading weights. When the animal had been placed in position under light anaesthesia weights were placed on the pan in increasing order and the weight just required to produce a fracture of the femur was noted.

Fracture thus produced was reduced by Closed Reduction Technique, set and plastered under general anaesthesia. Traction was given at the foot while one assistant fixed the upper part of the thigh. During the application of traction, the foot was kept at right angles to the longitudinal axis of the leg. The broken fragments were palpated, manipulated, and brought in alignment after avoiding angular or rotatory deformities. It was then plastered with the usual technique and checked under screen. The rats were examined periodically and checked radiologically.

Results

Results obtained in the three sets of cases are recorded in the three tables appended below. Table No. I shows that rats on a milk protein diet required much more weight to bring about a fracture of the femur than in the case of rats maintained on either gram or Bajra diet. Rats on Gram required more weight for fracture than on Bajra diet.

Union of fracture and the healing period was minimal in the case of rats on milk diet.

Table 1

Rats maintained on a balanced diet with milk as the chief source of protein

No.	Weight of the rat in grams	Weight required to produce fracture in grams	Number of days for joining	Condition of joint radiologically
1	65	1,840	6	+ + +
2	75	2,000	6	+ + +
3	80	2,000	6	+ + +
4	80	2,360	6	+ + +
5	80	2,360	6	+ + +
6	85	2,480	8	+ + +
7	90	2,360	6	+ + +
8	95	1,480	6	+ + +
9	110	2,480	6	+ + +
10	110	3,480	6	+ + +
11	115	4,000	5	+ + +
12	130	...	7	+ + +
13	140	4,000	4	+ + +
14	145	5,000	7	+ + +
15	145	4,500	8	+ + +
16	160	4,000	7	+ + +
17	165	5,000	8	+ + +
18	170	4,420	7	+ + +
19	180	5,720	6	+ + +
20	185	5,000	6	+ + +
21	185	2,720	7	+ + +
22	190	5.360	6	+ + +
23	195	4,480	8	+ + +
24	215	5,720		

Table 2

Rats maintained on a balanced diet with Gram as the chief source of protein

No.	Weight of animals in grams	Weight required to produce fracture in grams	Number of days required for joining	Condition of joint radiologically
1	40	1,360	6	+ + +
2	52	1,660	6	+ + +
3	53	1,660	6	+ + +
4	60	1,480	7	+ + +
5	65	1,000	8	+ + +
6	70	1,480	5	+ + +
7	80	2,240	8	+ + +
8	82	2,000	8	+ + +
9	85	1,480	8	+ + +
10	90	2,000	5	+ + +
11	90	1,720	8	+ + +
12	90	1,480	8	+ + +
13	95	1,480	7	+ + +
14	100	2,440	5	+ + +
15	100	1,720	8	+ + +
16	102	1,900	7	+ + +
17	110	1,840	8	+ + +
18	110	2,480	8	+ + +
19	110	1,840	7	+ + +
20	120	1,840	8	+ + +
21	120	2,360	8	+ + +
22	120	3,720	8	+ + +
23	165	3,360	7	+ + +
24	125	1,480	8	+ + +
25	130	1,480	7	+ + +
26	65	2,000	6	+ + +
27	65	3,900	8	+ + +
28	75	2,840	8	+ + +
29	80	2,360	8	+ + +
30	80	2,720	8	+ + +
31	80	2,480	8	+ + +
32	95	3,000	8	+ + +
33	95	2,180	9	+ + +
34	100	3,900	7	+ + +
35	100	2,900	10	+ + +
36	100	2,480	10	+ + +
37	105	2,600	7	+ + +
38	107	2,900	7	+ + +
39	110	2,480	10	+ + +
40	110	3,000	11	+ + +
41	115	2,660	7	+ + +
42	115	2,600	10	+ + +
43	115	3,000	8	+ + +
44	125	5,000	8	+ + +
45	130	3,240	10	+ + —
46	165	3,240	5	+ + +
47	235	7,480	8	+ + —

Table 3

Rats maintained on a balance diet with Bajra as the chief source of protein

No.	Weight of animal in grams	Weight required to produce fracture in grams	Number of days for joining	Condition of joint radiologically
1	70	1,180	8	— —
2	80	1,120	8	— —
3	85	1,120	8	— —
4	90	1,180	8	— —
5	95	2,000	8	— —
6	95	1,360	8	— —
7	105	1,360	8	— —
8	108	2,000	8	— —
9	110	1,360	8	— —
10	115	1,360	8	— —
11	115	1,420	8	— —
12	115	1,000	8	— —
13	115	1,240	8	— —
14	115	2,000	8	— —
15	115	1,300	8	— —
16	116	2,840	8	— —
17	120	1,000	8	— —
18	125	1,480	8	— —
19	130	1,450	8	— —
20	130	1,720	8	— —
21	140	1,600	8	— —
22	160	3,000	8	— —
23	170	3,000	8	— —
24	47	4,000	8	— —
25	90	5,000	8	— —
26	92	4,000	8	— —
27	107	4,000	8	— —

Practically all the rats had their fractures united radiologically within a period of 8 days. In the case of gram diet the period of union went upto 11 days, whereas union within 6-7 days was in a comparatively much smaller number of cases than in the case of milk fed animals where much larger number got their fractures united in six days. In the case of Bajra diet none of the rats had the fracture united in eight days.

Summary

1. Young healthy rats were divided into three batches. All of them were kept on a balanced diet except for the fact that the source of protein varied from batch to batch in the following order. Batch No. 1 was given milk as the chief source of protein, 2nd had grams and the third Bajra as the chief source of protein.

2. Fracture of the femur was produced by loading and the load required was noted.

3. Union of the fracture and healing was examined periodically and skiagram taken.

4. It was noted that the rats kept on milk as the chief source of protein required much more weight to produce a fracture than those on gram or Bajra diet. Those on gram diet required more weight for fracture than those on Bajra diet.

5. Rats kept on milk diet had their fractures united within a period of 6-8 days, a majority getting united in 6 days.

6. Rats kept on Gram diet required longer period, 6-11 days for the union, very few having the fracture united in 3 days.

7. Rats kept on Bajra diet did not have their fracture united in 8 days.

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MAFFUCCI'S SYNDROME AND DYSCHONDROPLASIA

Case Reports

BY

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Maffucci's Syndrome is a rare disorder, first described by Maffucci in 1881 as an association of Dyschondroplasia with Cavernous Haemangiomata and Phleboliths in the soft tissues. Carleton reviewed twenty cases reporting two of their own in 1942. Elkington, Robby Smith and Greenfield have also contributed papers regarding the same.

Various deformities of the skeleton occur. Dyschondroplasia being a rare one. It was first described by Ollier in 1900 as due to disturbance of bone formation at the growing ends of the bone. It is characterised by round masses or columns of cartilage in the metaphysis and diaphysis of certain bones. In this condition nests of cartilage become misplaced and persist in the metaphysis, the lesions being mainly endosteal in nature and not projections from the surface, as is seen in Aclasia.

Etiology of this condition is not known. Some factor is said to act in foetal life during the 4th and 6th months. (Speiser 1925) Due to the tendency of the columns of cartilage radiating towards the epiphyseal line from the entry site of the nutrient vessel, Benzton in 1924 pointed out that hyperaemia due to anomalies of Vegetative Nervous System may be responsible but experimental proof is lacking for the same. Hereditary and Familial influences do not seem to play a part in the disease. Males are more frequently affected than the Females.

The lesions in this condition show a marked tendency to unilateral distribution, occasionally involving a single bone. The disease affects the bones developed in cartilage, and at the more rapidly growing ends the lesions are mainly seen. In order of frequency of occurrence are the Knee Joint, Lower ends of Radius and Ulna, Upper ends of Femur and Humerus, Lower ends of Tibia and Fibula, and long bones of the Hands and Feet, particularly the Phalanges. Pelvis, Scapula, Ribs, Sternum, Skull and Tarsus are also affected. The Carpus and the Spine usually escape. The midpart of the shaft of a long bone is affected in most severe cases.

The clinical picture is characterised by extreme polymorphism. Usually the affected arm and leg are dwarfed to varying degree, sometimes being grossly affected resulting in shortening of limbs. Deformities like Genu Valgum and bending of Bones is observed when cartilage columns occupy one-half of the epiphysis. When bilateral, the growth lags. The enlargement of the hands due to large enchondromata is common, the hand becoming useless in severe cases. As in most affections of the skeleton, Ulna is shorter than Radius with bending of the shaft of the latter; the Fibula is also shorter than the Tibia, the latter usually not being bent as the Radius. Displacement of the Radial head, Cubitus Varus and Mannus Varus deformities are also reported. Facial asymmetry and involvement of the Cranial Nerves are seen very rarely. Pathological fractures are common with delayed and malunion occurring. Blood examination usually reveals no abnormality.

In radiological study, the affected metaphysis shows cartilage filled clear cystic areas of varying size and shape. The columns of cartilage as described earlier extend to a varying degree into the shaft. The metaphysis may be distorted in shape with enlargement which may be even gross. Fine striations may be seen in the metaphysis, these being columns of increased dense areas of bone between the cartilage columns. Breach in the cortex may be due to large masses of cartilage protruding through the cortex sometimes. Very rarely the whole shaft of a long bone may get involved producing mottled, cyst like changes causing much confusion in diagnosis. Irregular density of the epiphysis with mottling is seen in children; (5–10 years) sometimes it is being confined to one side of the epiphysis in which case the corresponding half of the metaphysis will be the seat of changes also. In the hands instead of columns of cartilage, multiple enchondromata are seen anywhere in the shaft, with at times loss of continuity of the crotex. In Ilium the columnar arrangement of cartilage will typically be seen radiating in a fan like fashion towards the Iliac crest. Circular dense spots in the

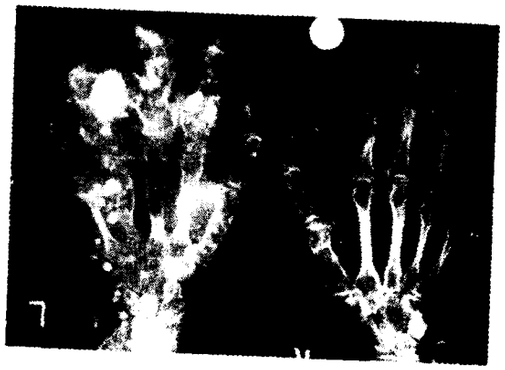

Fig. 1
A.P. view of right and left hands. Shows multiple enchondromata involving the metacarpals and phalanges of all digits in the left hand. In the right hand enchondromata are seen in the phalanges of the thumb, Index finger and in the fifth metacarpal bone. Phleboliths are seen in the left hand picture.

Fig. 2
A.P. and Lateral view of spine and lateral view of left forearm. Spine shows scoliosis with the convexity to the right in the Dorsolumbar region. The lateral view of forearm shows enchondromata at the lower ends of Radius and Ulna.

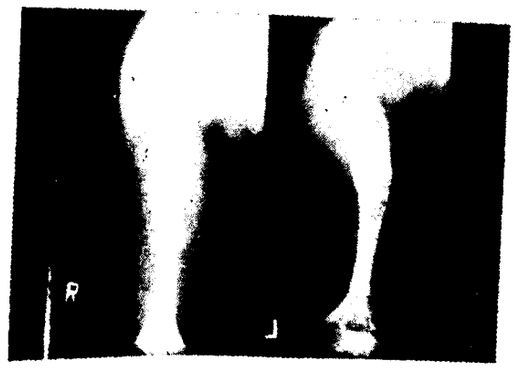

Fig. 3
A.P. of Right and left arm. Shows enchondromata in the upper end of left Humerus with bending of that part medially. The right Humerus is normal.

Fig. 4
A.P. View of Left Femur. Shows enchondromata at the upper and lower ends of the Femur and also in the inferior ramus of Pubis. Fracture of the shaft of Femur at the level of middle and lower 1/3 is also seen.

metaphysis are seen at times in adults indicating the healing stage of Dyschondroplasia.

The vascular anomalies usually consist of multiple Cavernous Haemangiomata with Phleboliths in the soft tissues. These may occur anywhere in the soft tissues. These form soft, reddish and at times tender tumours. When clacified they produce shadow in the radiographs. They are entirely independent of skeletal lesions, being purely coincidental and not in any way related to the other, in the causation or progress of the latter.

The misplaced cartilaginous rests proliferate usually in the hands and feet causing complete crippling of the hand at times. Chondrosarcomata develop rarely from these lesions.

Microscopically the masses consist of Hyaline Cartilage with cells of varying size, arranged in irregular manner with septa of fibrous tissue intervening. Calcification and Ossification is seen in elderly patients and degenerative changes in large enchondromata are reported. The Haemangiomata show large spaces lined by epithelium, and some of these get calcified.

Patient usually reports for deformities of limbs, swellings causing pressure on vital structures, pain, pathological fractures or very rarely after malignant changes have supervened. The affected persons are generally of low stature with poor muscular development and average intelligence.

The following two cases, one of Maffucci's Syndrome and another of Dyschondroplasia are presented because of their rarity.

CASE No. 1

Maffucci's Syndrome

Miss H, Anglo-Indian, aged 20 years.

Complaint : (1) Multiple painless swellings confined almost to the left side of the Body.

(2) Shortening of the Left Lower Limb.

Duration : Since birth.

History of Present illness : The patient remembered the swellings from the age of 7 years. The swellings were present in the region of the Left Hip, at the lower end of the Left Thigh, at the Upper and Lower ends of the Left Leg, Left Foot, at the upper end of the Left Arm, at the Lower end of the Left Forearm and the Left Hand. The right thumb and Index finger also showed the swellings. They were gradually growing in size, painless but interfering with movements, especially of the Left Hip, Left Knee and Left Hand. She observed shortening of the Left Lower Limb at the same age from which time she was compensating it by raising the Left Shoe for proper walking. Patient fell down due to slipping one year ago and sustained a fracture of the middle and lower 1/3 of shaft of Left Femur. She had treatment for the same in the hospital. Patient is now able to walk well with no pain at the fractured site.

Previous history : Nil particular. Mile stones in the development of the patient were normal when she was a child.

Family history : No member in her family suffered from a similar complaint.

Physical examination

General condition : Well nourished woman of 20 years, not anaemic. The left lower limb is shorter by 3" which was compensated by raised shoe for proper walking. *Multiple* swellings in the above described regions were present. Gait normal. There was Scoliosis with the convexity to the right at the Dorsolumbar region.

Description of swellings

Left Lower Limb : There was a swelling at the upper end of the thigh. Bony, irregular, painless, not pulsating and not tender. Movements of the left hip were limited mechanically due to the large swelling and were painless.

Swellings at the lower end of Femur and the upper ends of Tibia and Fibula have the same characteristics. Movements of the knee joint were also limited due to the same cause. The Tibia was bent anteriorily in the upper half. Clinical Photo (1). In the foot the first, second, and the third toes with the corresponding metatarsals appeared to be involved.

Left Upper Limb : Swellings with the same characteristics were found in the upper half of the Humerus which was bent medially, Lower ends of the Radius and Ulna and the whole of the Left Hand. Haemangiomata and Phleboliths were also felt in the Left Hand (Clinical Photo 2).

In the right hand the thumb and Index finger appeared to be involved.

Spine : There was scoliosis at the dorsolumbar region with the convexity to the right. No tenderness on any vertebra and no clinical evidence of involvement of any vertebra.

Other bones : Normal.

Other systems : Normal.

Investigations : Urine and Blood examination (Total and Differential Count, R.B.C. and Blood chemistry) revealed no abnormal findings.

X-Ray :—

1. *Hands :* Left Hand revealed multiple enchondromata involving the metacarpals and Phalanges of all digits. Phleboliths were also seen. In the right hand enchondromata were seen in the phalanges of the thumb, Index finger and in the fifth metacarpal bone.
2. *Forearm :* Enchondromata were seen at the lower ends of Radius and Ulna. Right forearm bones were normal. Spine shows scoliosis with the convexity to the right in the dorso-lumbar region.
3. *Arm :* Showed enchondramata in the upper half of the Humerus with bending of that part medially. Right arm bone was normal.
4. *Thigh :* Showed large sized echondramata at the upper and lower ends of Left Femur. Also showed a well united fracture of the

Fig. 5
A.P. and Lateral views of left Leg. Shows enchondromata at the upper and lower ends of Tibia and Fibula, with bending of the former anteriorly in the upper 1/3.

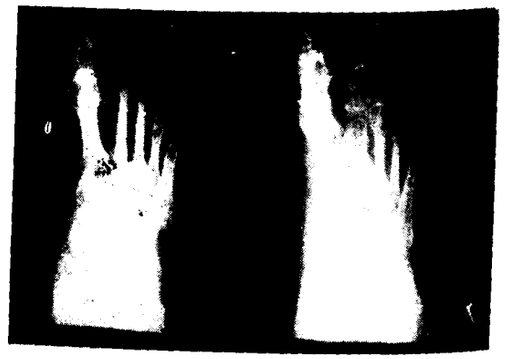

Fig. 6
A.P. View of Right and left Feet. Shows enchondromata in the phalanges of the first, second and third toes and the second metatarsal bone of left foot. The right foot bones are normal.

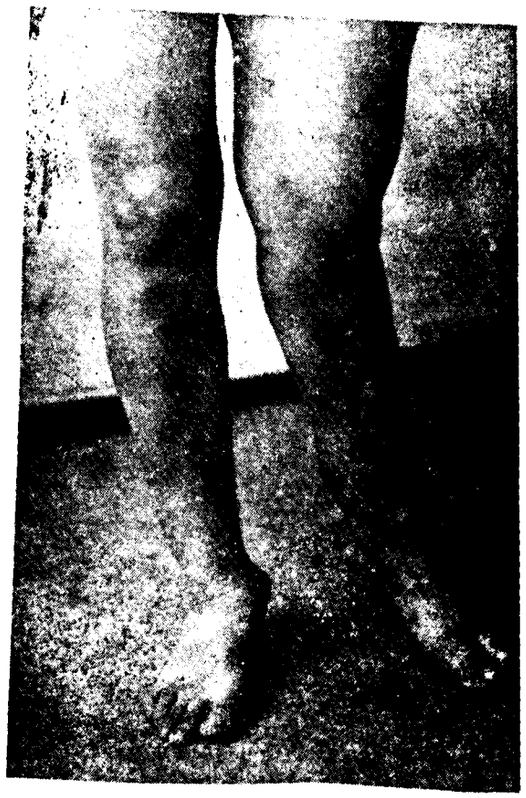

Fig. 7
Clinical photo of both lower limbs from the middle of thigh. Shows the shortened left lower limb and the Talipes Equinus Deformity of the Left Foot which is helpful in proper walking. Also shows swelling at the lower end of thigh and the leg.

Fig. 8
Clinical photo of left upper limb. Shows the deformed hand caused by multiple enchondromata of the phalanges and metacarpals. Also shows swelling at the lower end of forearm.

Fig. 9
Clinical photo of the patient. Front view. Shows the swelling at the upper end of right arm extending from the shoulder to about lower 1/3 of humerus. Also shows swellings at the right hand, upper and lower ends of right thigh, lower end of right leg and right foot. Also shows shortening of right upper and lower extremities.

Fig. 10
Clinical photo of the patient : Back view. Shows the same features as in the front view.

Fig. 11
Clinical photo of both the lower limbs. Front view. Shows the swelling at the lower end of thigh and leg. Also shows the shortened right lower limb. The swellings of the right hand can also be seen. The left limb is normal.

Fig. 12
Clinical photo of both the lower limbs. Back view. Shows the same features as in the front view.

shaft of Femur at junction of middle and lower 1/3. Right Femur was normal.

5. *Leg :* Showed enchondramata at the upper and lower ends of Left Tibia and Fibula, with bending of the former bone in its upper 1/3 anteriorly. Right Leg bones were normal.
6. *Foot :* Left foot showed involvement of the first, second, third toes, second and third metacarpals. Right foot bones were normal.

Fig. 13
A.P. and Lateral views of left knee joint. Shows enchondromata at the lower end of left femur and at the upper ends of Tibia and Fibula.

Comment : This is a typical case of Maffucci's Syndrome. Blood chemistry did not reveal any abnormal findings. The fracture of the shaft of Femur united well without complications like Malunion and Nonunion as reported in the literature. In spite of the deformed hand the patient was able to continue her job as Telephone Operator.

CASE NO. 2

Dyschondroplasia

Mr. M. N. Male aged 50 years.

Complaint : Pain in the right arm swelling and rapid growth of the same.

Duration : Since four months.

History of present illness : Patient said that he had swellings in the region of the right arm, right hand, upper and lower ends of right thigh, upper and lower ends of right leg and right foot since birth. There was shortening of the right upper limb by 2″ and the right lower limb by 3″. The swelling of the right arm had begun to increase rapidly in size causing pain since four months. Patient came for the above ailment and was not worried about the other swellings.

History of past illness : Nil particular. Mile stones of the development of the patient were normal when he was a child.

Family history : No other member in his family had similar complaint.

Occupation : Patient is a labourer by occupation. Earns daily wages by preparing and selling baskets. Patient was able to continue his work despite the swellings in the right hand.

Physical examination

General condition : Moderately nourished individual of 50 years. Swellings in the above described regions were present. The right upper limb was shorter by 2″ and the lower by 3″. Patient was able to walk but with a limp to the right side and was also able to do his work.

Descriptions of swellings : The swelling of the right arm was large in size extending from the shoulder region to about the lower 1/3 of the Humerus. Skin was glossy, no dilated veins, no pulsations, bony hard, not warm, Tender Chondroma of the upper end of Humerus. Movements of the shoulder joint were limited due to the mechnical obstruction of the swelling. Pain and rapid growth were perhaps due to the malignant change in the swelling.

The right hand showed swellings involving all digits and 2nd and 3rd metacarpals. They were painless, bony hard and not tender. Enchondramata of the Metacarpals and Phalanges. They were interfering with the movements of the fingers. Muscle power was good.

Swellings with the same characteristics as that of the right hand were present at the upper and lower ends of right Femur, upper and lower ends of the right Tibia and Fibula, First and Second Metatarsals, and in the Great toe. Movements of the right Hip and Knee were present in full range and painless. The swellings were moderate in size.

Other bones : Nil particular.

Other systems : Nil particular.

Investigations : Urine and Blood examination revealed no abnormal findings.

X-Ray :

1. *A.P. view of Right Shoulder :* Showed large enchondramata at the upper end of Right end of Right Humerus with malignant changes supervening.
2. *A.P. and Lateral Views of Right Hand :* Showed enchondromata involving all digits and second and third metacarpals.

Patient refused Disarticulation of shoulder which was suggested for malignancy.

Comment : This is a typical case of Ollier's Disease with multiple chondromata involving the right half of the body. The rapid growth and pain and the swelling of the right arm were due to malignant changes in the tumour. Patient was able to pursue his job in spite of the deformed right hand and was also able to walk well (with a limp to the right) without pain. Blood chemistry did not reveal any abnormal findings.

ACKNOWLEDGEMENT

My thanks are due to Dr. V. R. T. Swamy, M.S., F.R.C.S., M.Ch. (Orth) (Eng.) ex-professor of the Orthopaedics and the present D.M.S. of Madras State for permitting me to report these cases.

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LATERAL ENCEPHALOCELE

BY

R. SARASWATHY AMMAL, TRIVANDRUM (S. INDIA) *

Spina bifida, meningocele, or meningo-encephalocele occurring in the middle line of the occipital region are not uncommon. But congenital hernial Protrusions of intra-cranial contents through openings in the skull are rare. Ian Aird (1957) quoting Rathke (1839) Breschet (1826) says that such congenital protrusions may occur as encephalocele in relation with Foramen Caecum, Cranio-pharyngeal canal and present as such in the nose or on the lateral side of the skull. Following is a case report of a lateral encephalocele.

A well-nourished female child 30 days old was admitted to the Hospital for a swelling on the right side of the face and neck. The swelling was present from birth. It was about the size of an egg and gradually grew in size. It was a soft cystic swelling located on the right side extending from the tragus to the angle of the mouth horizontally; and from the zygoma to the Thyroid cartilage vertically. (Fig. 1). The overlying skin showed prominent veins. The cyst was fluctuant, non-pulsatile, irreducible and brilliantly translucent. There were no signs or symptoms referable to the nerves or any cranial defect. Other systems showed no abnormality. The cyst was suspected to be a cystic Hygroma and excision of the cyst was done. Post-operative period was uneventful and the child was discharged with wound completely healed.

The excised cyst measures 7.5 × 5.5 × 5 cms. Wall was a few mm thick, whitish and congested and lobulated. On section it contained clear fluid and showed an inner lining formed by a thin bluish membrane which was attached to the outer one with thin connecting bands. The inner surface of the cyst was trabeculated. (Figs. 2 and 3).

Histologically the cyst was lined by cuboidal cells resembling the ependymal cells of the ventricle. (Fig. 4). The substance of the wall showed congested cortical brain tissue. In some places nerve fibres were seen lying outside this layer. (Figs. 5 and 6).

Fig. 1
Diagram showing the site of lateral encephalocele on the right side in a child aged 30 days.

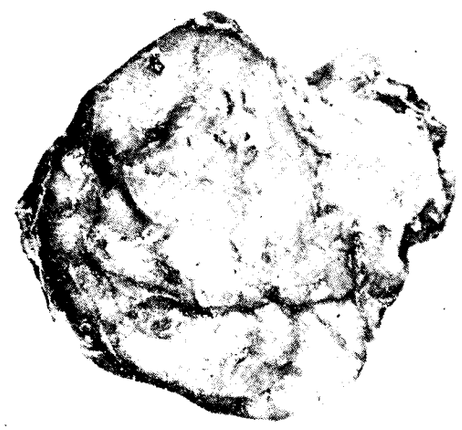

Fig. 2
Specimen of Lateral Encephalocele, outer surface showing lobulation.

*From the Department of Pathology, Medical College, Trivandrum.

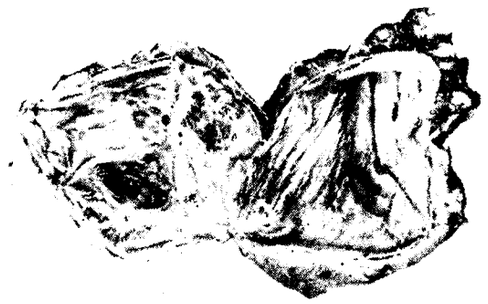

Fig. 3
Section of the encephalocele, the inner surface of the cyst showing trabeculation.

Fig. 5
The section shows cortical brain tissue. (×450).

Fig. 4
Microscopic structure of the wall of the encephalocele showing the three layers—ependymal lining, cortical brain tissue and nerve fibres. (× 100).

Fig. 6
Section shows the inner [illegible]al lining of the encephaloc[illegible]).

Discussion

Zachariae (1953) gives incidence of Encephalocele as occurring one in 3000–5000 of new born infants. The defects of the skull through which brain protrudes vary in size and are seen most often in the midline especially at the base of the skull involving the squamous portion of the occipital bone. Anterior and lateral defects are very uncommon and hence encephaloceles in such locations are rare.

In the case reported no cranial defect was discovered during operation or on clinical examination and there was no connection between this tumour mass and the brain by nervous on meningeal tissue. In a series of human embryos from 7.5 to 30 mm. which belonged to the collection of the Michigan University Patten (1952) observed a curious distortion of the Central Nervous System which was described as "overgrowth of the Neural tube". It appeared that in several embryos the neural tube epithelium had started to grow widely, so that it became folded and refolded on itself, as it became crowded into a cranial space fairly normal in size and shape. In some cases the redundant brain had caused the head to become high crowned or as one might expect, the brain to break through the overlying tissues resulting in an Encephalocele. This type of overgrowth and encephalocele was noted in a series of early embryos but not in embryos at later stages of pregnancy or at birth, the cause of which is not definitely known. If such overgrowth is the factor producing the anomaly in this case and persisted throughout the development, it is easy to explain the origin of this encephalocele. When the cranium developed from the mesodermal elements perhaps this part became devoid of its connection with the rest of the brain and ventricles. The increase in the size of the swelling may be attributed to the production of fluid by the cells similar to the ependymal cells lining the ventricle and due to accumulation of the same because of no drainage. The translucency can be explained by the fact that there was only a thin layer of brain tissue around, in the wall of the cyst.

Summary

1. A report of a rare case of lateral encephalocele has been made.

2. The embryological aspects concerned in the development of such an anomaly is explained.

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ASSOCIATION NOTES

THE XX ANNUAL CONFERENCE

The XX Annual Conference of the Association of Surgeons of India will be held at Visakhapatnam on the 27th, 28th, 29th and 30th December, 1958. The following Papers will be discussed :—

1. **Acquired Facial Defects**
   by Dr. M. Mukherji, Calcutta
2. **Hydronephrosis—Experimental and Clinical Studies**
   by Dr. B. N. Balakrishna Rao, Gwalior & Dr. A. E. deSa', Bombay
3. **Cysts of the Lung**
   (i) Hydatid Cyst of the Lung—
   by Dr. S. K. Sen, New Delhi
   (ii) Cystic Disease of the Lung—
   by Dr. G. S. Karai, Calcutta

Besides this, a few 'Short Papers' will also be read at the Conference. Members desirous of presenting short papers at the Conference are requested to inform the Hony. Secretary before the 15th of September, 1958 of their intention to do so and enclose a short precis of the paper at the same time. The time allotted for each paper is 20 minutes.

As the papers have to be circulated to a panel of referees who will decide regarding the acceptance or otherwise of the papers, members are specially requested to send in their complete papers (in triplicate) before the 15th of October, 1958. Papers received after the 15th October 1958 will not be accepted.

Subjects for the Main Scientific discussions at the XXIII Annual Conference in December, 1961

The 23rd Annual Conference of the Association of Surgeons of India will be held during December 1961. Three subjects for the main scientific discussions at the above Conference will have to be selected by the General Body at its annual meeting at Visakhapatnam in December this year. Members are requested to send in the titles of their subjects, if any, so as to reach the Hony. Secretary not later than 30th of November 1958.

Subjects for Discussion at future meetings

21st Meeting :

1. **Place of Vascular grafts in Obliterative Vascular diseases**
   by Dr. R. Nigam, Nagpur & Dr. V. V. DaSilva, Bombay
2. **Uses of Radio-active Isotopes in Surgery**
   by Dr. Miss P. Parvathi, Calcutta & Dr. S. R. Mukherjee, Calcutta

22nd Meeting :

1. **Reconstructive Surgery of the Biliary Tract**
   by Dr. S. J. Mehta, Bombay
2. **Clinical & Experimental Studies in Keloids**
   by Dr. K. K. Ghosh and J. L. Sinha.

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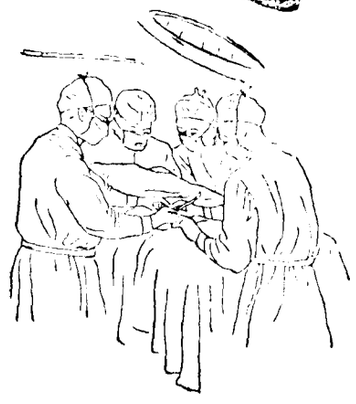

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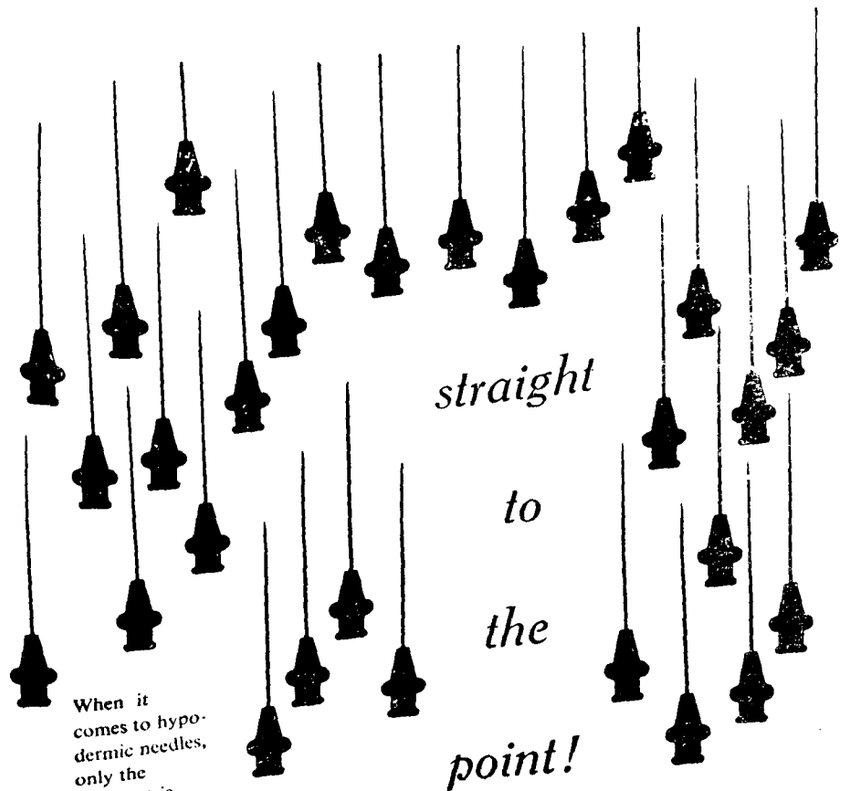

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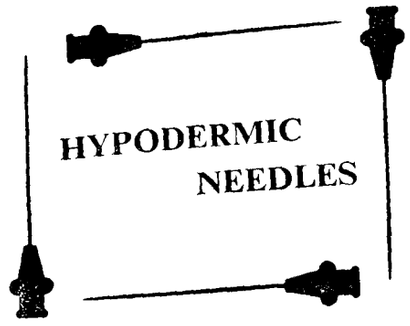

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