Clinicopathological Conference

A Case of Giant Follicular Lymphoma with Radiation Nephritis

DEMONSTRATED AT THE ROYAL POSTGRADUATE MEDICAL SCHOOL

Clinical History

Dr. O. M. Wrong: The patient (Case No. 18813; P.M. No. 11786) was a housewife aged 43 at her death in 1966. The first symptoms of her main illness were noticed in 1955, when she developed generalized pruritus and crops of boil. Later that year she noticed swelling of the left side of the abdomen and in early 1956 a mass on the left side of the neck. On admission to Hammersmith Hospital she was found to have enlarged lymph nodes in both sides of the neck and axillae and a deeply placed irregular lump 7 cm. in diameter to the left of the umbilicus. The spleen and liver could not be felt. A full blood count, bone marrow examination, and plasma protein electrophoresis were all normal. The Wasserman and Kahn reactions were positive, but the Treponema pallidum immobilization test was negative. A chest radiograph showed her to have enlarged hilar nodes. A biopsy was taken of a lymph node in the left axilla, but both this and a lymph node removed in another hospital before admission here were then considered to show non-specific changes.

On clinical grounds she was thought to have giant follicular lymphoma, and she was treated with chlorambucil, which led to a reduction in the size of the nodes. In 1957 her cervical nodes were once more enlarged, and she was therefore given x-ray therapy (skin dose 1,800 r) to both sides of the neck and the nodes diminished in size but the pruritus persisted and the enlarged hilar nodes remained. In 1958 her abdominal mass was larger, and she therefore received x-ray therapy to the abdomen (skin dose 1,230 r). The skin irritation and the masses disappeared, and for the next three years she was very fit.

In December 1963 she developed bilateral ankle oedema, a recurrence of lumps at all the previous sites, and large nodes in the iliac fossa; a chest x-ray showed a recurrence of her hilar nodes and atelectasis at the left base. A barium meal showed that the stomach was displaced anteriorly, suggesting the presence of a retrogastric mass. The blood urea was 28 mg./100 ml, and the urine and bone marrow were normal. She was given a further dose of radiation to the whole abdomen (1,700 r tumour dose).

Her symptoms did not improve, and in May 1964 she was readmitted to hospital with increased oedema and dyspnoea, tenderness over the sternum, spine, and lower ribs, bilateral pleural effusions, and enlarged nodes in the neck and axillae. Her blood pressure was recorded as 120/80 mm. Hg. The haemoglobin was only 8.3 g./100 ml., the white blood count was 5,000/cu. mm., and the Coombs test was negative. Fluid (350 ml.) was aspirated from the left pleural space and did not contain any obvious tumour cells. The blood urea was now 55 mg./100 ml., but she had no proteinuria. A chest radiograph showed bilateral effusions, enlarged nodes in the mediastinum, and a collapsed left lower lobe. Treatment with chlorambucil was once more given (for six days only) and prednisone 15 mg./day was started. After this treatment her bone pain disappeared.

Further Progress

Over the next two years she remained fairly well, but required local radiotherapy for masses in the neck, both axillae, and the right chest wall. Prednisone was continued. In April 1966 she developed new symptoms: blurring of vision in the left eye, retrosternal pain on exertion, and oedema of the eyelids. She complained of thirst and frequent vomiting, nocturia, and a loss of weight of 13 kg. during the previous year. In hospital she was found to have papilloedema, a blood pressure of 200/110 mm. Hg, crepitations at both lung bases, an enlarged heart with a pericardial friction rub, bilateral ankle oedema, and perichaeia over the legs. Her urine contained up to 7 g. of protein per day. The blood film showed occasional contracted red cells and fragments. The haemoglobin had fallen to 7.1 g./100 ml., the white blood count to 1,700-3,600/cu. mm., and the platelets were 30,000-130,000/cu. mm. The blood urea had risen to 285 mg./100 ml., with potassium 3.0-3.8, and bicarbonate 27-35 mEq/l. Serum electrophoresis showed the serum albumin to be 2.9 and globulin 2.8 g./100 ml., with a low gamma and raised alpha 1 globulin. An x-ray film of the abdomen showed symmetrical kidneys which were slightly reduced in size.

Treatment for hypertension was commenced with bethanidine and methyldopa, and she was transfused with blood and given a 20 g. protein diet. Her blood pressure was gradually controlled in the 100-110 mm. Hg diastolic range, and her eyesight improved. The blood urea fell slightly at first, but then became stabilized around 255 mg./100 ml. The serum gamma globulins were estimated as gamma G 350, gamma A 90, and gamma M 25 mg./100 ml. On 5 June she was discharged home slightly improved—only to be readmitted 10 days later with a recurrence of pulmonary oedema, a blood pressure of 170/110 mm. Hg, a raised jugular venous pressure, and a urine output of under 400 ml./day. Despite diuretics her weight rose, and peritoneal dialysis was therefore commenced.

While on dialysis the patient's daily urine output dropped further to under 200 ml./day, even though the blood urea remained high; dialysis was therefore continued. Late in June she complained of blurred vision again, and physical examination showed a variable lower quadrant right homonymous hemianopia, for which there was no apparent cause. On 7 July treatment with azathioprine (Imuran) 50 mg./day was commenced, as a decision had been taken to attempt renal transplantation. There was no change in the peripheral blood picture. On 11 August a cadaveric kidney of the correct blood type became available and this was inserted into the patient's right iliac fossa by Professor R. Slackman and his colleagues. The operation was attended by an unprecedented oozing of blood, and the patient was transfused with 15 units of blood during the procedure. She was given high corticosteroid cover usual for this operation, and azathioprine was continued 100 mg./day.

After her operation the patient never did well. About 12 hours after returning from theatre her blood pressure fell to 60 mm. Hg systolic and she lost consciousness. She may have inhaled vomit; certainly a blood-stained fluid was sucked out of her bronchial tree. With artificial ventilation her blood pressure recovered, but she never regained complete consciousness. No more than a few drops of urine were passed post-operatively, and frequent haemodialysis was necessary. She developed generalized purpura and melaena. After 21 August...
Clinical Diagnosis

(1) Disseminated lymphoma, ? giant follicular.
(2) Chronic leucopenia and thrombocytopenia, due to therapy and the underlying lymphoma.
(3) Renal failure with malignant hypertension, probably the result of radiation nephritis.
(4) Renal homotransplantation, with tubular necrosis in transplant.
(5) Final coma due to either cerebral haemorrhage, septicaemic or anoxic brain damage.
(6) Probable acute peptic ulcer.
(7) Terminal bronchopneumonia.

Post-mortem Findings

Professor C. V. Harrison: The body was that of an emaciated middle-aged woman weighing about 45 kg. and 157 cm. in height. There were numerous petechial spots all over the body, more marked peripherally than centrally, and there was the incision from the recent operation in the right iliac fossa with stab wounds close by. It was also a tracheostomy wound, cutdown incisions in the arms and the ankles, and a superficial pressure sore over the sacrum.

Sections of the original 1956 biopsy were available by courtesy of Dr. Jane Fullerton of St. Olave’s Hospital. These showed a giant follicular lymphoma of the lymph nodes of a well-differentiated lymphocytic type. In retrospect, our failure to recognize this in 1956 was a diagnostic error. At necropsy the lymph nodes were small and difficult to find. In the neck no lymph nodes were found but only fibrous scars. In the axilla very small nodes were found, and those in the mediastinum were about normal size. The abdominal nodes were small and fibrous, and none could be found in either the iliac or inguinal regions. Microscopically all the nodes were completely free from tumour. There was some scarring. There was gross depletion of lymphoid tissue and some evidence of a histiocytic sinus reaction. The spleen (320 g.) was about twice normal size. It was free from any sign of lymphoma and also showed considerable depletion of normal lymphoid tissue. It was not fibrosed, and the extra weight appears to have been due to a simple increase in the pulp. The bone marrow appeared to be red, but on microscopic examination the colour was found to be due to numerous blood vessels and there was, in fact, gross depletion of all bone marrow elements. There was no sign of any lymphoma.

The pericardium was normal. The heart weighed 380 g. (normal 260 g.). The increased weight was entirely due to left ventricular hypertrophy, the left ventricle being 20 mm. thick instead of 15 mm. The coronary arteries showed only minor fibrous intimal thickening. These, with the aorta, showed only isolated atheromatous plaques.

The patient’s own kidneys were small, the left weighing 110 and the right 93 g. (normal = 150 g.). The length of the kidneys were 105 mm. left, and 90 mm. right. The kidneys were uniformly shrunken, the colour rather red, and the cortex considerably reduced. Microscopically the striking lesion was atrophy of the cortical tubules with interstitial fibrosis. The glomeruli were patent, though a minority were fibrosed. Some glomerular tufts showed foci of fibrosis and very rarely points of necrosis. The arteries at all levels showed remarkably severe lesions. The lumina were reduced by very great intimal thickening. There was hyaline change and occasional necrosis in the arterioles. The arterial changes by themselves could possibly have been the result of malignant hypertension, but taken with the tubular destruction in the cortex the whole picture strongly indicated radiation nephritis (Figs. 1 and 2).

The renal transplant lay in the right iliac fossa. The three anastomoses, artery, vein, and ureter, were all healthy and intact. The kidney measured 145 mm. in length and weighed 302 g. It appeared swollen and the cortico-medullary junctions were slightly blurred. Microscopically the kidney showed interstitial oedema but there were no signs of parenchymatous or vascular rejection. There was, however, evidence of tubular damage and there were casts in the distal renal tubules. These appearances suggested simple tubular necrosis following temporary ischaemia and were probably recoverable (Fig. 3). The renal arteries showed fibroelastosis of some degree, but not excessive for the age of the donor. There was a small, simple cortical adenoma at the lower pole of the left kidney.

The peritoneum showed fibrinous peritonitis with small flocks of fibrin scattered over the peritoneal cavity and occasional adhesions. Microscopically many of these flocks proved to be colonies of fungus, apparently Candida albicans (Fig. 4). There was relatively little inflammatory reaction. There was a small acute duodenal ulcer.

There was severe tracheitis below the tracheostomy opening. The left lung showed bronchopneumonia, and, in the upper lobe, a rather larger area of consolidation. Microscopically these lesions proved to be fungus infection due to candida. The right lung also showed bronchopneumonia, less well defined than in the left. Gram-negative bacilli were seen in section and klebsiella was grown from cultures.

The meninges were normal. In the brain the left subcortical white matter just posterior to the motor area contained a 15 mm. focus of discoloration and posterior to this a few smaller foci of apparent early necrosis. Microscopically these also proved to be foci of candida infection with minimal cell reaction (Fig. 5). The ovaries and the endometrium were atrophic, presumably due to an irradiation-induced menopause.

Pathologist’s Diagnosis

(1) Giant follicular lymphoma, irradiated, cured.
(2) Radiation nephritis with malignant hypertension and renal failure.
(3) Renal transplantation with tubular necrosis and temporary functional failure.
(4) Peritonitis due to Candida albicans with septicaemia and systemic candidiasis.
(5) Brain abscess due to candida.
(6) Bone marrow failure with leukaemia and thrombopenia.
(7) Acute duodenal ulcer.

Discussion

Dr. Wrong: This patient received quite enough radiation to develop radiation nephritis. It was not widely recognized at the time that that amount of radiation could cause renal damage. The largest series of cases of radiation nephritis that I know of is from the Christie Hospital in Manchester and was collected by R. A. Luxton. These were patients with testicular seminoma who received radiation to the paraaortic glands. Luxton’s conclusions were that any dose over 2,300 r could cause radiation nephritis, and from this one must conclude that our patient received enough radiation to develop this complication. I think if one was in this situation again one would argue that the kidneys should be shielded from the abdominal radiation, or perhaps the radiation should be omitted altogether because the lymphoma was not a very malignant one. Perhaps the abdominal condition should have been treated with cytotoxic drugs only.
FIG. 1.—Irradiated kidney. Loss of tubules and replacement fibrosis. Glomeruli recognizable but crowded. (H. and E. ×61.)

FIG. 2.—Irradiated kidney. Arcuate artery showing almost complete occlusion by intimal fibrosis. (Elastic van Gieson. ×160.)

FIG. 3.—Transplanted kidney. Interstitial oedema separating the tubules. Protein casts and cell debris fill some of the tubules. Interlobular artery and two glomeruli are healthy. (H. and E. ×114.)
Clinicopathological Conference

Fig. 4.—Granule on peritoneal surface consisting of a colony of fungus (Candida). (Grocott. \( \times 141 \).)

Fig. 5.—Cerebral cortex with a sulcus at bottom and edge of necrotic lesion showing fungal hyphae. (H. and E. \( \times 56 \).)
This was a young, healthy-looking woman who enjoyed life. Perhaps in retrospect we should not have submitted her to renal replacement. An alternative was the possibility of the patient developing chronic renal insufficiency, but the thought of her being on to the dialysis programme was distressing.

Professor C. C. BOOTH: Thank you very much, Dr. Wrong. This is an extremely interesting story. Let us start with the lymphoma. Professor Harrison, am I correct that the lymphoma was cured? There was no sign of it at necropsy.

Professor C. V. HARRISON: This is difficult to answer. In the simple, anatomical sense that we searched from the base of the skull down to the pelvis and we failed to find any tumour, that is true. Whether some malignant cells are still lurking unseen and would have blown up in another year or two, that is speculation.

Professor BOOTH: Have you any comments on the lymphoma? It is always said that Brill's disease is a much more benign condition than Hodgkin's. Do all the figures bear that out?

Professor HARRISON: This is so tiresome. Most of the good prognoses are due to inaccurate diagnoses. A large number of patients who have done extremely well for years may have been inaccurately diagnosed by the pathologist. That's one big difficulty. The second point, which comes out in the paper of Rappaport and others, is that follicular lymphoma is not one single condition. There are varieties or grades included in this diagnosis. At one end of the scale there is the follicular lymphoma in which the cells are anaplastic. Patients with this disease die relatively quickly. At the opposite end of the scale are the patients in whom the tumour cells are very well differentiated and these people do very well. Our patient's histology fits with this latter group.

Professor BOOTH: The usual treatment for this is abdominal radiotherapy, since the presentation of this particular type is often with multiple masses in the abdomen, so much so that the abdomen often feels like a sack of potatoes. In this case the therapy very effectively relieved the patient of her lymphoma, and we go on from there to what the radiotherapy produced—namely, the renal lesion. How often does this happen?

Incidence of Radiation Nephritis

Dr. WRONG: I don't think anyone has done a prospective investigation. What has been done is to delineate people who have got this form of renal failure after radiotherapy and to find out how much radiation they had had and how long it had taken them to get renal failure. Patients may develop an acute radiation nephritis, which if the radiation has not been very great may improve with the passage of time. However, the late development of renal failure, very frequently accompanied by malignant hypertension, can occur at intervals of 5–10 years after radiation. This patient, judging from what I've read, was fairly typical.

Dr. D. EVANS: Surely most of these people die of lymphoma before they develop radiation nephritis. The dose given is of the usual order for abdominal lymphoma. Luxton's work was done in patients treated for seminoma. Though the hazard of radiation nephritis is known, it is generally accepted that the prognosis of abdominal lymphomas is bad, and radiotherapy is often given as a palliative measure rather than as a cure.

I would like to ask whether an estimate is available for the kidney dose, because the dosage here seems to approach the lower limit known to cause radiation nephritis. On the first occasion the skin dose was 1,230 r, and the kidney and tumour doses presumably were lower. The tumour dose was 1,700 r on the second occasion and the kidney dose presumably lower. Even if you add both the doses together, this makes only 2,900 r, and this is in a split dose rather than continuous doses over 40 days, which is the usual therapy.

Dr. WRONG: I am just not competent to discuss this. From discussing this matter with our radiotherapists I gather that it is not really justifiable to add up the radiation dose taken at two different times and say that this is equivalent to the same total dose given all at once.

Professor BOOTH: I think we have to accept from Professor Harrison's data that it is radiation nephritis and that obviously the dose here was enough.

Cause of the Hypertension

Dr. J. HOBB: Have you envisaged the mechanism of the hypertension of the radiation nephritis? If you believe that angiotensin is responsible then it is of interest that in the sections shown there were very few juxtaglomerular apparatuses to make any.

Dr. L. BEILIN: One could envisage that this patient's kidney had multiple small obstructions to the arterioles leading to the radiation damage to the vessels which had caused fibrosis around them. The relationship between renal hypertension and angiotensin is not clearly understood. There is a very close relationship between salt balance and renin production but not necessarily a very good correlation between renin and blood pressure in established cases of human renal artery stenosis, for example; so the lack of renal tissue capable of leading to angiotensin production doesn't rule out the possibility of this being renal hypertension. It's equally possible that the radiation destroyed some anti-hypertensive factor produced by the kidneys.

Professor BOOTH: Would you like to comment on the response to therapy?

Dr. BEILIN: The therapy seemed quite appropriate as far as I could see. She'd been treated with traditional anti-hypertensive drugs—clearly diuretics could not be used.

Dr. E. KOHNER: Could I just say that she still had the papilloedema from her hypertension just before the renal transplantation?

Dr. WRONG: Yes, she had deteriorated in the very few days that she was out, and she still had papilloedema.

Professor BOOTH: So, despite treatment, she had not responded very well.

Dr. WRONG: However, there wasn't very much time. I know that papilloedema will disappear with treatment of hypertension, but she came to us on 11 May, and her final admission was on 15 June. Papilloedema does take a little time to disappear.

Dr. KOHNER: About four weeks.

Dr. J. G. AZZOPARDI: Before we get off the renin question, Dr. Beilin, renin isn't produced in the tubules, is it?

Dr. BEILIN: No, it's thought to be produced in the juxtaglomerular apparatus.

Dr. AZZOPARDI: The juxtaglomerular apparatus is situated between the afferent arteriole and the distal convoluted tubule. Dr. J. HOBBS: But there didn't seem to be much there either.

[Discussion continues with further medical details and clinical observations.]
many mild forms of hypertension associated with renal artery stenosis the plasma renin level is normal.

Dr. Wrong: The hypertension associated with radiation nephritis has some rather strange features. Professor C. Wilson has found that hypertension can result from radiation nephritis in an animal with an exteriorized kidney without histological abnormality in the kidney. Another thing which he and his colleagues have definitely established is that hypertensive damage to the kidney on the opposite side from the renal artery stenosis in the experimental animal renders the kidney more prone to damage from radiation.

Professor Booth: The problem is whether the rat kidney behaved in the same way, and this has not yet been proved.

Dr. Wrong: It is quite likely that radiation renal damage is worse in a patient who’s got basic hypertension or an underlying glomerulonephritis.

Professor Booth: Transplantation clearly would have been an ideal method of treatment if the transplant had worked. There was evidence at necropsy of tubular necrosis and this is one of the early causes of renal failure.

Professor Harrison: But recoverable.

Professor Booth: Supposing she had come through, would she have recovered?

Dr. Wrong: I think she would have recovered renal function. Nowadays more and more cadavers are being used for renal transplantation and it is recognized that the transplanted kidney will often develop tubular necrosis. The period of renal ischaemia is much longer than when a kidney from a live donor is used. One must be able to support the patient by dialysis until the transplant recovers. Almost everyone who is doing renal transplantation now is prepared to do this.

Professor Booth: So this kidney could have recovered if she’d gone on. The problem was that she developed an infection. One thing that I didn’t notice here was the antibiotic therapy that she’d had.

Antibiotic Cover

Dr. Wrong: She did receive a number of antibiotics, but I suspect she had no antibiotics to cover the transplantation. She was under the care of Professor Shackman and his team, and in general their views are very much the same as our own—that is, not to treat an infection until it occurs and then to use the appropriate antibiotic. I see here notes about polymyxin, methicillin, ampicillin, and kanamycin. She was on corticosteroids all the way through—she’d had a small dose before operation as part of the treatment for her lymphoma, but this was increased to 300 mg. of prednisone for the first three days after the transplantation.

Professor Booth: So she’d been on corticosteroids and multiple antibiotic therapy, which is the correct treatment in this situation. Dr. Gorbach, would you like to comment on the candidiasis?

Dr. S. Gorbach: This patient had many of the factors which predispose to candida infection. Firstly, she had the remnant of atypical lymphoma; then she received the course of corticosteroids and immunosuppressive drugs, and finally a wide variety of antibiotics. I wonder, specifically, if she was receiving tetracycline, because this is one agent which predisposes to candidiasis in animals.

Dr. Wrong: I can’t find a record of tetracycline in these charts.

Haematology

Dr. M. Brain: May I comment about the haematology. I doubt if the chronic leukaemia and thrombocytopenia were due to radiotherapy. I have examined her serial blood counts, and in fact she did not become leukopenic until some considerable time after her radiotherapy. Indeed, in May she had a total white cell count of 8,000/cu. mm and this raises the question of other factors causing a fall in leukocytes and platelets. The bone marrow shown by Professor Harrison was taken after azathioprine had been given, and one should be cautious in interpreting these late bone marrow findings in attempting to explain changes before it had been given. Could this patient have developed folic acid deficiency? She was on a very restricted diet and also underwent prolonged peritoneal dialysis. Folic acid deficiency might explain both the leukaopenia and the thrombocytopenia. This raises a further point. Though the usual regimen after renal transplantation is to give patients azathioprine and corticosteroids in large doses, I wonder were we not just adding a further unnecessary insult? She may already have been in a state of immunological depression from the underlying lymphoma and leukaemia. Might she not have been managed without either the azathioprine or the large dose of corticosteroids?

Dr. Wrong: She did receive multiple vitamin supplements after her renal transplant.

Dr. C. Pallis: There are two points of neurological interest. The first is her initial referral to the neurological clinic in May 1966. This woman with lymphoma was thought by the radiotherapists to have developed raised intracranial pressure from intracranial deposits. She had headaches, vomiting, and a severe retinopathy. Closer clinical examination revealed evidence of renal failure and malignant hypertension, and she was referred to Dr. Wrong.

The second point concerns her terminal illness. Neurologically this really consisted of two illnesses. The first was a massive cerebral anoxic insult, sustained during the operation or immediately after it, the cause of which is not immediately apparent. She remained in a postoperative coma for 10 or 15 days without showing any clearly lateralized neurological signs. I suspect that more detailed examination of the brain would show changes of cerebral anoxia in the appropriate areas. While she was slowly improving from this episode I think she developed a septicemic illness from candida resulting in the formation of a cerebral abscess. The clinical evidence of this second neurological illness is the late onset of hemiplegia.

Professor Booth: The crucial point here is that this lady died of a candida septicaemia which was not recognized in life, and the question is how it should have been and how it could have been.

Dr. Gorbach: It is extremely difficult in a patient who has received many antibiotics to decide whether she has acute candida infection. There are cultures in which in these circumstances will grow fungi, but this by itself is not sufficient. If, however, the sputum culture grows a pure culture of candida to the virtual exclusion of other micro-organisms this adds a little more weight. If a patient with a pulmonary infection fails to respond despite various manipulations with the antibiotic regimen this should arouse suspicion that the infection is non-bacterial and possibly fungal. Finally, frequent blood cultures are needed to isolate organisms from the blood stream.

Professor Booth: How often do you do that in a patient with candida septicaemia? How often do you get it in the blood cultures?

Dr. Gorbach: You get it fairly often if you have taken cultures frequently in the initial febrile period. The problem is what to do when there is a positive candida blood culture. In this patient nothing much could have been done except to stop the antibiotics in the hope that she would replace her normal flora. The only effective treatment is amphotericin B and there was no chance of giving this to cure her because of the severe renal disease.

Dr. K. Weinbren: Has anyone tried hyperbaric oxygen for candidiasis?

Dr. Gorbach: This fungus will grow with or without oxygen, or at high oxygen tensions—it’s very hard indeed.
Dr. M. K. WALLACE: We have seen severe candida infection in the peritoneum of another patient who came to one of these meetings about 18 months ago—a patient of Professor Fraser’s—and it is of interest, I think, that she also had had immunosuppressive drugs. While doing routine cultures of peritoneal fluid on all patients who are on peritoneal dialysis we have turned up some candida infections, and these patients have had vomiting and some abdominal pain. They have been treated on two or three occasions very efficiently with amphotericin B in a small intraperitoneal dose in terms of clearing the culture and remission of the vomiting.

Dr. HOBBS: One point I would stress is that although she may have had defective humoral immunity the main defence to candidiasis is thought to be cellular immunity. Defective cellular immunity is present right at the beginning of these diseases and does not respond to treatment as far as we know.

Professor BOOTH: Unfortunately, despite heroic efforts, this lady died. I think we'll end there.

We are grateful to Professor J. P. Shillingford and Dr. E. D. Williams for assistance in preparing this report, and to Mr. W. Brackenbury for the photomicrographs.

REFERENCES

NEW APPLIANCES

Sternal Plate for Repair of Pectus Excavatum

Mr. H. H. BRADMORE, F.R.C.S., thoracic surgeon, St. Mary's General Hospital, Portsmouth, writes: Although the effect of pectus excavatum on respiratory and cardiac function may not be pronounced, operative repair in severe cases is justifiable for cosmetic reasons. The basis of operative treatment is complete liberation of the deformed lower sternum and correction of its position by performing an osteotomy at the junction of normal and deformed bone. It has been shown that late relapse after maintenance of the corrected position by suture at the osteotomy is almost invariable (Moghissi, 1964). A number of external and internal methods of fixation have been described; among the latter are metal struts (Adkins and Blades, 1961), Steinmann-type pins (Mayo and Long, 1962), and wire mesh (May, 1961). A strut or plate appears to be the most effective method of maintaining postoperative correction; it should have the following properties: (1) a large and accurate bearing area both on the posterior surface of the sternum in the median plane and on the ribs and intercostal structures laterally; (2) it should be of thin section, consistent with adequate strength, to allow close apposition and easy removal, and shaped to provide slight overcorrection; and (3) it should be composed of non-reactive material.

A plate with these features is shown in Fig. 1; the central part is slightly convex anteriorly, it is tapered to facilitate removal, and it is provided with terminal holes for fixation to the costal cartilages and intercostal muscles by suture. Three sizes are available.

After exposure of the deformed sternum by a transverse incision and reflection of the pectoralis and rectus attachments it is mobilized by separation of the xiphoid and by wedge chondrolysis of the deformed cartilages, preserving the perichondrium. Radical resection of cartilage is not required, and results in weak fibrous regeneration. Correction is completed by a transverse wedge osteotomy of the sternum, and the bone is freed from its posterior attachments.

The plate is passed behind the sternum below the osteotomy and sutured in position (Fig. 2), the sternal attachments of the pectoralis and rectus muscles are refixed, and the retrosternal space is drained.

In view of the marked tendency to recurrence in this condition the plate should not be removed before six months in children; in adults it may be left indefinitely. When removal is indicated one end of the plate is exposed by a short incision and grasped with bone-holding forceps. It can then be easily slid out from behind the sternum.

The plate has been in use for the past four years, with results that appear satisfactory from the viewpoint of freedom from recurrence, easy removal, and absence of wound infection.

The plates are made in 18-gauge titanium by Messrs. Down Bros., to whom I am indebted for their co-operation.

REFERENCES