tion for men, but not as strikingly as bronchogenic carcinomas.

On the basis of histologic and clinical evidence, the scar was thought to be tuberculous in four cases (3,5,8,13). In the remaining cases the data were not conclusive, but a healed infarct (cases 9,12,2,10), granulomatous disease (4,11), or nonspecific organized pneumonia (1) were considered to be the cause of the scarring.

The role that the scar might have played in the pathogenesis of the neoplastic process is obscure. Some believe that the atypical epithelial proliferation and, in a further step, malignancy, are the results of an abortive effort of the damaged epithelium to regenerate, thus rendering it more susceptible to neoplastic processes.8 Others have suggested the possibility of carcinogenic influence of foreign substances trapped in the scar, as anthracotic pigment or carcinogens (chemicals or viruses) adsorbed on the inhaled particles of carbon or dust.12 Berkheiser15 has also discussed the role of tissue hypoxia in this context.

Another possibility is that the "restless" epithelium in areas of tissue repair is more susceptible to neoplastic evolution or to carcinogetic agents. In conclusion, the scarring may play a dual role in the pathogenesis of these tumors, by causing an increased concentration of potentially carcinogenic agents and by stimulating the epithelium to regenerate, thus rendering it more susceptible to the latter.

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REFERENCES
1 Friedrich, G: Periphere Lungenkrebsé auf dem Boden pleuranaher Narben. Virch Arch Path Anat 204:230, 1939
9 Yokoo H: Peripheral lung cancers arising in scars, Cancer 14:1205, 1961
13 Zatuchni J, Campbell WN, Zarafonetis CJD: Pulmonary fibrosis and terminal bronchiolar (alveolar-cell) carcinoma in scleroderma. Cancer 8:1147, 1953

Anomalous Collateral Systemic Pulmonary Circulation to a Normal Lung

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A case of anomalous systemic pulmonary collateral circulation is reported. The left lung, which was somewhat smaller than normal but did not otherwise show any abnormalities, was perfused almost completely with arterial blood derived through precapillary anastomoses from a huge collateral network. The latter was formed by the lateral thoracic, intercostal and internal mammary artery. No apparent reason for the development of this collateral circulation was found.

Anomalous collateral systemic-pulmonary circulation has been well documented in chronic lung disease1-3 pleural adhesions,4 congenital heart disease,5,6 isolated absence or malformation of one of the pulmonary arteries7-11 and in veno-occlusive disease of the pulmonary veins.12 Accessory pulmonary arteries derived from the systemic circulation to part of a lung, with otherwise normal bronchovascular connections, have repeatedly been described.13-15 Massive systemic blood supply through collateral vessels to a lung with a normal bronchial tree and a nonobstructed pulmonary artery

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CASE REPORT

A 56-year-old woman schoolteacher was admitted to the hospital for further investigation of unilateral rib notching, found on a routine chest-x-ray examination. According to the patient this anomaly existed for at least 30 years. During childhood, she had an acute pleuropulmonary disorder, for which hospitalization was not required. The exact nature of this disease was unknown, but she recovered completely. The patient complained of a mild chronic nonproductive cough for 20 years.

On physical examination no abnormalities were found except the presence of palpable arterial pulsations and a moderately intense continuous murmur in the left axillary and lateral thoracic region. The arterial pressure was 14/8 cm Hg.

The results of blood analysis were: hemoglobin, 14.5 gm per 100 ml; hematocrit, 39.5 percent; white blood cell count, 5,100/mm³; sedimentation rate (Westergren), 5 mm after one hour; BUN, 24 mg per 100 ml; blood glucose, 84 mg per 100 ml; serum cholesterol, 245 mg per 100 ml. The result of urine analysis was completely normal.

A 14 lead electrocardiogram revealed only slight flattening of the T wave and mild ST segment depression in lead I, V4, V5, V6 and V7, and was otherwise normal.

A chest x-ray film (Fig 1) showed marked notching of the fourth to eighth left ribs. The left lung was somewhat smaller than normal and the left diaphragm was moderately elevated, but no herniation of the right lung to the left side was seen. The left pulmonary artery and its branches were clearly present. In the left costophrenic region a poorly delineated opacity was noted. On fluoroscopy the left diaphragm showed normal respiratory movements and no mediastinal swing could be detected.

Scanning of the lungs after intravenous injection of macroaggregates of human 131I albumin revealed a normal distribution of the tracer over the right lung; practically no tracer could be detected over the lower portion of the left lung, but over the upper part of this lung a moderate amount was seen (Fig 2).

The vital capacity of the lungs was 2,256 ml and the one second forced expiratory volume 1,908 ml, respectively 80.2 percent and 83.1 percent of the expected values. At rest the arterial oxygen saturation was 98 percent, the Pco₂ 36 mm Hg and the pH 7.45 units; after a 10-minute period of exercise on a bicycle ergometer in the supine position at a workload of 100 watts, the arterial oxygen saturation was 98 percent, the Pco₂ 34.5 mm Hg and the pH 7.47 units.

A bronchogram of the left lung showed a completely normal bronchial tree without any deformation or displacement.

A combined right and left heart catheterization was performed. The catheter could easily be manipulated not only in the right but also in the left pulmonary artery and its branches to the lower and upper lobes. Table 1 shows the pressure values and oxygen saturation at different levels of

| Table 1—Results of Right and Left Heart Catheterisation |
|-----------------|-----------------|-----------------|-----------------|
|                 | Oxygen Saturation, % | Pressure, mm Hg |                 |
|                 | Syst. | Diast. | Mean |                 |
| SVC             | 68    |       |      |                 |
| RA              | 68    |       |      |                 |
| RV              | 69    | 24    | 5    | 3               |
| MPA             | 70.5  | 25    | 11   | 15              |
| RPA             | 70.5  | 25    | 11   | 15              |
| LPA             |       |       |      |                 |
| UL              | 81    | 25    | 11   | 15              |
| LL              | 98    | 25    | 11   | 15              |
| PC              |       |       |      | 9               |
| Aorta           | 98    | 170   | 80   | 120             |
| LV              | 98    | 170   | 10   |                 |

SVC: superior vena cava; RA: right atrium; RV: right ventricle; MPA: main stem of the pulmonary artery; RPA: right pulmonary artery; LPA: left pulmonary artery; UL: upper lobe of the left lung; LL: lower lobe of the left lung; PC: pulmonary capillary position; LV: left ventricle.
An oxygen saturation step of ten percent was found in the left pulmonary artery and the blood was completely arterialized in the branches of the left pulmonary artery supplying the lower lobe. The total left to left shunt was estimated by an indirect indicator dilution method. Blood was sampled through a Waters densitometer cuvette at a rate of 30 cm³ per minute consecutively from the femoral and pulmonary artery. Cardiogreen was injected into the superior vena cava. The time interval between each injection was approximately two to three minutes. The cardiac output was calculated by the Stewart-Hamilton principle. Table 2 shows the consecutive outputs of the left and right ventricle respectively. The calculated mean left to left shunt was 1.10 liter per minute.

Selective pulmonary angiocardiogram was performed by injecting 60 cm³ meglumine diatrizoate (Urografin) into the main stem of the pulmonary artery. The left pulmonary artery was visualized somewhat more slowly, its branches were sparser, had smaller diameter and were less filled with contrast material than on the right side. Branches to the paracardiac region of the left lung were not visualized. Injection of 50 cm³ of Urografin 76 percent selectively in the left pulmonary artery also showed no visualization of the left paracardiac branches, except for their origin. Thereafter 60 cm³ Urografin 76 percent was injected into the arch of the aorta and this revealed opacification of a larger than normal left subclavian artery. The left lateral thoracic artery was as large as the brachial artery, had a tortuous course and communicated through the thoracic wall with a huge network of collateral vessels, which had developed over the whole left lung except the apex. The left mammary and intercostal arteries very likely contributed also to this network (Fig 5). On later films, the left pulmonary artery, including the left paracardiac branches, were clearly visualized (Fig 6).

**COMMENTS**

In the present case, an extensive transpleural collateral circulation to the left lung, originating from the left lateral thoracic artery and very likely also from the internal mammary and intercostal arteries, was demonstrated.

It is not known if the bronchial arteries participated in
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the collateral circulation. The definite oxygen saturation step up in the left pulmonary artery and its marked filling after injection of contrast material in the arch of the aorta, indicate the presence of precapillary anastomoses with the systemic collateral vessels and marked backflow of oxygenated blood from the smaller ramifications to the larger branches. The precapillary admixture of arterialized blood was most marked in the paracardiac branches of the left lower lobe. Completely arterialized blood was sampled from this region and the backflow branches of the left lower lobe. Completely arterialized blood entered the left lung. Some minor differences existed between the present and the two previously reported cases. Selective pulmonary arteriography failed to visualize the pulmonary artery of the affected lung in both cases of Tammeling and co-workers and only in case 3 the catheter entered this vessel. The exact source of the collateral circulation was also not demonstrated in both these cases.

REFERENCES

2 Liebow AA, Hales MR, Lindskog GE: Enlargement of the bronchial arteries and their anastomoses with the pulmonary veins had a normal appearance on the selective pulmonary arteriography. Although the left diaphragm was moderately elevated and the left lung appeared to be somewhat smaller than normal on the chest x-ray film, there was no reason to suspect organizing parenchymal lung disease. Indeed, the bronchogram of the left lung was completely normal and there was no arterial desaturation or carbon dioxide retention at rest or after exercise. Massive systemic-pulmonary arterial shunt through multiple pleural adhesions has been described. It is unlikely that in the present case important pleural adhesions were present since the left diaphragm showed normal respiratory movements on fluoroscopy.

From the foregoing discussion it appears that in the present case a considerable amount of blood was shunted from the systemic circulation to an apparently normal lung through an extensive collateral circulation. No one of the usual causes responsible for a systemic pulmonary collateral circulation could be identified. A survey of the literature revealed the extreme rarity of this condition. We found only two cases (Tammeling and co-workers, case 1 and 3) which are similar to our case. In both these cases and in the present case the alveolar-capillary bed of one lung, which is smaller than normal and has a practically normal bronchial tree, is perfused with arterial blood derived from systemic-pulmonary collateral vessels. Bronchospirometric studies in Tammeling’s cases 1 and 3 showed that the oxygen uptake on the affected side was practically zero and that only arterialized blood perfused the lung. Since we did not perform bronchospirometry in our case, it is impossible to be sure that only completely oxygenated blood entered the left lung. Scanning of the lung after intravenous injection of macroaggregates of human I131 albumin, however, suggests strongly that only a small quantity of venous blood entered the left lung. Some minor differences existed between the present and the two previously reported cases. Selective pulmonary arteriography failed to visualize the pulmonary artery of the affected lung in both cases of Tammeling and co-workers and only in case 3 the catheter entered this vessel. The exact source of the collateral circulation was also not demonstrated in both these cases.

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Carcinoma of the Esophagus Engrafted on Lye Stricture*

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A 34-year-old Negro woman developed progressive dysphagia approximately 12 years after ingestion of lye. At this time, she was unable to swallow liquids, weighed 72 pounds, and complained of constant chest pain. She was found to have extensive carcinoma in the lower third of the esophagus, which was resected. A causative relationship between the lye stricture and the carcinoma is suspected.

The association of lye stricture and carcinoma of the esophagus has been known ever since Cattell1 presented such a case to the Pathological Society of Philadelphia in 1896. It appears that association of these two conditions in his case was a coincidence, as the interval between the lye ingestion and development of carcinoma was extremely short. He stimulated interest in this subject, however, and several reports have appeared in the literature since, which indicates that there may be a cause and effect relation between these two conditions.

In this report such a patient is described and a brief review of literature is given.

**Case Report**

A 34-year-old Negro woman ingested lye by accident in June, 1956. She recovered from the acute stage without receiving any specific treatment. Other than mild dysphagia, she did relatively well until three months prior to this admission to the hospital when she started experiencing progressive dysphagia which did not respond to dilatation. By the time she was admitted to the Thoracic Surgery Service of the City of Memphis Hospitals on February 1, 1969, she was unable to swallow even liquids (Fig 1). She weighed 72 lbs and complained of constant substernal pain. Following hydration and blood transfusion, a feeding jejunostomy was performed on February 12, 1969. Approximately three weeks later, after she had gained 10 lbs, thoracotomy was performed. At surgery on March 17, 1969, she was found to have extensive carcinoma involving the lower third of esophagus and extending to the stomach and descending thoracic aorta. A palliative esophagogastrectomy with primary esophagogastrostomy was performed. She made an uneventful recovery and was discharged on April 2, 1969. Histologic sections showed poorly differentiated squamous cell carcinoma of esophagus. The prognosis was poor.

**Discussion**

Benedict2 reviewed 16 cases from the literature of carcinoma of the esophagus engrafted on lye stricture and added one of his own. Reviewing 381 patients with esophageal corrosion, Kiviranta3 reported eight such cases. Three additional cases were reported by Arrants

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