the "better patient selection" excuse for defining the future role of skeletal muscle assistance.

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Aortopulmonary paraganglioma, a rare tumor

To the Editor:

I read with interest the letter on aortopulmonary paraganglioma by Castañon and associates,¹ who referred also to our case of intrathoracic chemodectoma (or paraganglioma) with multiple localizations.² I would like to recall the Carney triad of extraadrenal paraganglioma, pulmonary chondroma, and gastrointestinal leiomyo(sarco)ma. After reviewing the clinical data and the available histologic data on our case, Carney included our case in a series of patients with the triad from the Mayo Clinic and from the literature.³

Our case concerned a female patient who underwent at the age of 31 years a resection of an anterosuperior mediastinal chemodectoma. An esophageal anomaly had been discovered at the age of 16 years, during an episode of vomiting, but because the patient was afterward asymptomatic, she refused any further investigation of the esophagus. Seventeen years later the patient had a second operation. An aortopulmonary chemodectoma was resected and a pulmonary osteochondroma of the lingula was enucleated. The esophagus was not explored according to the wish of the patient. Two years later a small tumor in the left upper lung lobe was visible and was considered to be a new or recurrent benign pulmonary hamartochondroma, but again 2 years later an inoperable squamous cell carcinoma of the left main bronchus was discovered and the patient died 3 months later at the age of 52 years. Postmortem examination was refused by the relatives, so that the histologic type of the esophageal tumor is still unknown. We believed that the long-lasting

esophageal anomaly was a benign leiomyoma and considered that our patient had the Carney triad.

With regard to the case of aortopulmonary paraganglioma reported by Castañon's group,¹ I have some recommendations and some comments.

I recommend regular follow-up of the patient, exclusion of other localizations of the paraganglioma, and exclusion of the triad of Carney. There is no chronologic sequence in the appearance of these three tumors, and their occurrence is more than coincidence.³ Moreover, malignancy of each part of the triad is possible.^{2,3}

The risk of lung cancer is increased in patients with chondromatous hamartoma, whether the hamartoma itself or the fibrous scar or whether both tumors have common risk factors.⁴ The spatial association was present in our case, and the question arises whether the enucleation of a hamartoma should not be altered in a segmental resection. In our case a new small tumor appeared 2 years after the enucleation and was, without proof, considered to be benign, but another 2 years later lung cancer was discovered in the same area.

Finally, patients with pulmonary hamartoma have a high incidence of associated anomalies and benign tumors but also an increased susceptibility to development of malignant tumors. Therefore, the clinical significance of pulmonary hamartoma is in the workup and lifelong follow-up of those patients.⁵

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12/8/55995

Anomalous origin of the left coronary artery from the pulmonary artery: New technique or modification of "old" technique

To the Editor:

We read with interest the article by Tashiro and associates,¹ "Anomalous Origin of the Left Coronary Artery

Fig. 1.

From the Pulmonary Artery: New Operative Technique." Use of native pulmonary artery to fashion a tube that would then allow connection of the left coronary artery to the aorta is an excellent option when direct implantation is not possible.

We discovered a photograph in one of our slide collections (J.A.W.) that depicted an operation proposed by Dr. Harris Shumacker at Indiana University in 1962, more than 30 years ago (Fig. 1). Although the infant died before the operation could be undertaken, it was performed in the autopsy room.² We believe this "old" technique should be in the congenital heart surgeons' armamentarium when faced with a patient in whom the left coronary artery cannot be directly reimplanted and some other technique is required to span the distance from the aorta to the ostium of the left coronary artery.

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Topical effect of aprotinin on the surgical wound in cardiac surgery

To the Editor:

The best way to apply aprotinin is unknown in cardiac surgery, because the mechanism of aprotinin in preserving hemostasis remains under investigation.^{1,2} We have been recommending the use of low-dose aprotinin (2 million KIU) in the pump prime, because we demonstrated that low-dose aprotinin is sufficient to preserve platelet glycoprotein Ib receptors³ and ristocetin agglutination capacity of platelets⁴ during cardiopulmonary bypass (CPB). These seem to be the main mechanisms of aprotinin in preserving hemostasis. This efficacy and the sufficiency of low-dose aprotinin were reproduced by the platelet aggregation model on the extracellular matrix.⁵ Another possible mechanism of aprotinin in preserving hemostasis is the preservation of fibrin hemostatic sealing by the inhibition of fibrinolysis.¹ However, the direct effect of fibrinolysis in impairing hemostasis remains unclear,¹ because no clear evidence has been collected regarding how much the increase of fibrin-split products in the systemic circulation stands for the lysis of fibrin hemostatic sealing on the wound surface.

Of great interest is a recent report of Tatar and associates,⁶ who demonstrated the efficacy of aprotinin (1 million KIU) on hemostasis when used topically in the pericardial cavity before closure of the thorax cavity. This observation certainly suggests that the inhibition of fibrinolysis on the closed thorax cavity stabilizes the fibrin hemostatic sealing on the wound surface, contributing to hemostasis. If aprotinin is applied topically, its effect seems solely based on antifibrinolysis. In a previous study, we⁷ demonstrated enormous activation of fibrinolysis on the surgical wound by sampling blood from the thoracic cavity. This method might reveal the topical effect of