Surgical Treatment of Congenital Cardiovascular Diseases

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Many types of congenital cardiac lesions in dogs require cardiac catheterization for definitive diagnosis and cardiopulmonary bypass for corrective surgery. Because of practical limitations, this chapter is restricted to lesions and surgical procedures in which cardiopulmonary bypass is not required and in which the need for cardiac catheterization is minimal. These lesions include patent ductus arteriosus, persistent right aortic arch, pulmonic stenosis and tetralogy of Fallot. Together, these account for about 60 per cent of the cases of congenital cardiovascular disease seen in a canine hospital clinic.

Thoracotomy through the left fourth intercostal space without rib resection provides adequate exposure for nearly all the operations for these lesions in dogs, as well as for the removal of heartworms. All thoracic operations in dogs require positive pressure ventilation and preferably inhalation anesthesia to facilitate more rapid recovery and the return of spontaneous respirations.

**PATENT DUCTUS ARTERIOSUS**

Patent ductus arteriosus (PDA) is the most commonly diagnosed canine congenital cardiovascular anomaly and the one most frequently corrected surgically (Fig. 1).
The classification of PDA as a congenital anomaly is perhaps open to slight argument, since it is normally present at birth and becomes a lesion when it fails to close for unknown reasons. A series of 23 operated cases included more poodles (7) and German shepherds (5) than other breeds of dogs. In a larger series including the 23 operated cases just mentioned and nonoperated cases, the higher incidence in poodles was statistically significant and suggests that genetic factors may be implicated. Uterine environmental factors may also play an etiologic role, as evidenced by frequent occurrence of PDA in human infants following maternal rubella infection in the first trimester of pregnancy.

The ductus arteriosus is a normal vascular structure in fetal circulation that shunts pulmonary arterial blood into the aorta, thus bypassing the nonfunctioning lungs. Within hours after birth, this vessel normally constricts and functionally is closed. Anatomic closure is normally achieved within the first three weeks of life, and the resulting structure is called the ligamentum arteriosum. If the ductus arteriosus remains patent, blood continues to flow through it but in the opposite direction of fetal flow. This is because pulmonary artery pressure decreases after birth owing to inflation of the lungs. At the same time, systemic vasoconstriction and other factors increase peripheral resistance, which raises aortic blood pressure even higher above the decreasing pulmonary arterial pressure. Thus, ordinarily, the blood flow through the PDA after birth begins from the aorta to the pulmonary artery.

The cardinal diagnostic sign of an operable PDA is the presence of a continuous (machinery) murmur over the left precordium. Other clinical features are discussed in the preceding section (see Congenital Heart Disease p. 83).

In dogs with patent ductus arteriosus, enlargement of the left side of the heart with or without mitral valve thickening may cause coexisting mitral insufficiency due to anular dilatation. This is usually not detected until after the PDA has been corrected. In some instances, the heart size later decreases normally and the mitral insufficiency murmur disappears. In others, the heart shadow remains considerably enlarged and mitral insufficiency persists. It is likely that more severe valvular thickening with organic mitral insufficiency is present in these cases.

In dogs with patent ductus arteriosus, congestive heart failure, mainly of the left side but occasionally generalized, has been seen as early as three months and as late as seven years of age. In most instances, however, marked cardiac enlargement and congestive heart failure occur before three years of age. Since these factors adversely affect the prognosis for successful surgery and a normal postoperative life span, it is recommended that surgical correction be done as soon as possible after the diagnosis of PDA with left-to-right shunt. If signs of congestive heart failure are present, surgery should be postponed until the maximum effect of treatment with digitalis, diuretics and easter rest has been obtained. In some instances it is not possible to completely eliminate the signs of congestive heart failure. In such cases, it was necessary to compensate during several days of therapy, surgical correction resulted in elimination of the failure.

Dogs with marked cardiac enlargement should also be digitalized preoperatively, even though overt signs of congestive heart failure are not evident. It is also wise to avoid whole blood transfusions and usual amounts of intravenous fluids during surgery, unless they are considered absolutely necessary. If blood transfusions are deemed essential, it is advisable to give packed red blood cells rather than whole blood, to avoid precipitating pulmonary edema.

**Surgical Principles and Techniques**

The aim of surgery for this condition is to stop blood flow through the PDA. This may be achieved by ligation of the abnormal vessel or by division and suture between two oculding clamps. Ligation is easier to accomplish and is an acceptable method when the ductus is narrow, cylindrical and long enough to permit tying at least two ligatures far enough apart so they do not have a tendency to slide together. In these cases an additional transfixing suture-ligature should be placed between the two circumferential ligatures. In practice, all dogs with PDA have this type.

In most dogs, the width of the ductus is greater than the external length and the ductus is wedge shaped, so that it is necessary to slide to the narrowest portion of the ductus adjacent to the pulmonary artery. The ligature is placed as close as possible to the pulmonary artery without permitting return of flow through the ductus. It is advisable to perform a ligature in these cases, because in some cases recurrence of flow through the ductus have been observed. It is, therefore, advisable to divide and oversew the ductus. In dogs to divide and oversew the ductus. In dogs to divide and oversew the ductus. In dogs to divide and oversew the ductus. In dogs to divide and oversew the ductus. In dogs to divide and oversew the ductus. In dogs to divide and oversew the ductus. In dogs to divide and oversew the ductus. In dogs to divide and oversew the ductus. In dogs to divide and oversew the ductus. In dogs to divide and oversew the ductus. In dogs to divide and oversew the ductus. In dogs to divide and oversee the ductus.

The thorax ordinarily is entered through a left fourth intercostal space incision, taking dorsi muscle and ventrally just to the costochondral junction. In dogs with marked cardiac and pulmonary artery enlargement the fifth interspace provides better exposure. After initiation of positive pressure ventilation, the fourth and fifth ribs are spread as far as possible, and the pericardium and cardiac lung lobes are reflected inside the thorax. The PDA can be seen or palpated under the pleura between the aorta and the pulmonary artery at the level of its junction. It is located where a line drawn through the long axis of the main pulmonary artery intersects the left vagus nerve (Fig. 6). Before dissecting around the ductus, a 1- to 2-cm. opening should be made in the pericardium just ventral to the left vagus nerve, caudal to the root of the pulmonary artery (opposite the transcardial sinus). This permits easier clamping of both the aorta and pulmonary artery through the transverse sinus if a major vessel is inaccessible at this time. As an additional precaution, an umbilical trap is often placed around the ductus to stop retrograde blood flow in the event of an emergency. In lieu of a trap, two small incisions can be made in the mediastinum, dorsal and ventral to the descending aorta, to permit quick application of a vascular cross clamp (never modified hemostat).

Dissection of the PDA is begun with an incision in the mediastinal pleural, dorsal and parallel to the left vagus nerve.
and the mitral insufficiency murmur. In others, the heart shadow is considerably enlarged and mitral insufficiency persists. It is likely that myocardial thickening with organic mitral regurgitation is present in these cases.

With patent ductus arteriosus, congestive heart failure, mainly of the left side, has been seen from three months and as late as 2 years of age. In most instances, however, cardiac enlargement and congestive heart failure occur before three years of age. In these cases, surgical treatment may be advisable after the diagnosis of PDA has been obtained. In some instances, it is impossible to completely eliminate the congestive heart failure. In two dogs, congestive heart failure was evident on radiographic examination, surgery should be performed until the maximum effect of diuretics and digitalis was achieved. In cases of marked cardiac enlargement, ductus arteriosus can usually be isolated along the critical medial aspect of the failure. In two cases, surgery was successful after opening the pericardial sac near the origins of the brachiocephalic trunk and the left subclavian artery. In the opposite direction, it extends a similar distance caudal to the PDA. Usually, an easily dissectible plane is found between the pleura and the adventitia of the aorta. Dissection is best accomplished with blunt-tipped, curved, small Metzenbaum scissors, used to cut strands of tissue that come under tension when traction is applied in an outward direction with fine, blunt-tipped thumb forceps. The dissection plane is followed toward the ventral surface of the aorta, cranial and caudal to the ductus as well as around the ductus itself.

Extreme caution is required in dissecting the cranial aspect of the ductus. In this area, the aorta, right pulmonary artery and ductus are in immediate apposition, often thin-walled and hyperpulsatile. These factors, coupled with poor visibility, make it easy to puncture one of these vessels in an area where repair is nearly impossible.

A long PDA (1 cm. or more) can usually be isolated along the critical medial aspect by extrapericardial dissection with ventral displacement of the ductus. In these cases, the aorta, right pulmonary artery and ductus are in immediate apposition, often thin-walled and hyperpulsatile. These factors, coupled with poor visibility, make it easy to puncture one of these vessels in an area where repair is nearly impossible. A long PDA (1 cm. or more) can usually be dissected along the cranioventral aspect of the ductus. In this area, the aorta, right pulmonary artery and ductus are in immediate apposition, often thin-walled and hyperpulsatile. These factors, coupled with poor visibility, make it easy to puncture one of these vessels in an area where repair is nearly impossible. A long PDA (1 cm. or more) can usually be dissected along the cranioventral aspect of the ductus. In this area, the aorta, right pulmonary artery and ductus are in immediate apposition, often thin-walled and hyperpulsatile. These factors, coupled with poor visibility, make it easy to puncture one of these vessels in an area where repair is nearly impossible. A long PDA (1 cm. or more) can usually be dissected along the cranioventral aspect of the ductus. In this area, the aorta, right pulmonary artery and ductus are in immediate apposition, often thin-walled and hyperpulsatile. These factors, coupled with poor visibility, make it easy to puncture one of these vessels in an area where repair is nearly impossible.
dorsal pericardial reflection, until there is sufficient room and visibility to dissect cranially and caudally around the ductus.

In some dogs with a very short, window-type ductus, it is impossible to complete the caudomedial dissection without transecting small branches of the left recurrent laryngeal nerve, and occasionally, the entire nerve. This has been done without evidence of adverse postoperative effect other than changing the tone of one dog's bark.

After completely freeing the PDA from adjacent structures along its entire length, it is either ligated or divided. If the ductus is cylindrical and more than 1 cm. long, a ligature of 0 or No. 1 silk is tied at each end. An additional crisscrossing suture-ligature of 4-0 cardiovascular silk on a fine needle is tied in the center.

In the more common type of PDA, the vessel is wedge or funnel shaped, being narrowest adjacent to the pulmonary artery. This type should be divided if at all possible to avoid recurrence of blood flow. A straight or angled vascular clamp is positioned transversely on the ductus and crowded next to the pulmonary artery before being closed slightly more than enough to stop blood flow through the ductus. This can be determined by the disappearance of the thrill in the main pulmonary artery. At no time should this or any other vascular clamp be allowed to rest or hang freely on a vessel. They should always be held gently and every effort made to avoid a twisting action on the vessel wall. A second vascular clamp is similarly placed next to the aorta, leaving as much tissue as possible between the two clamps. If less than a 4-mm. segment of the ductus remains, another clamp is placed behind the pulmonary artery clamp, even if it includes more of the wall of the pulmonary artery. Care must be taken to avoid completely occluding the pulmonary artery and also to be certain that the distal tip of the second pulmonary artery clamp is still exposed and not attached medially to the wall of the right pulmonary artery. The initial clamp on the pulmonary artery side of the ductus is now carefully removed. After rechecking both remaining vascular clamps to be certain no portion of the ductus appears to be nonoccluded, the ductus is divided.

To divide the ductus, a very small incision is made midway in the lateral fold of severed ends of the ductus to prevent it from being pulled together. If any blood is getting interrubbing of sutures on opposing sides, the supposedly occluded segment. If so, then to aid in establishing firm blood clots, the nonoccluding clamp (determined by the needle holes) is opened another notch diurn is now closed, and if there is still hemorrhage, the ductus is now completely transected, intact pleura to permit suturing is used a scalpel or very sharp, straight scissors ductus area without undue tension. Care should be taken to avoid sutureting is done. However, if this causes a cut edge. If less than a desirable amount of vascular or nerve tissue, of the ductus is present between the oc-ductus area is left exposed. An incision including clamps, the division is made off cemen made in the ventral mediastinum, so that more is left on the aortic side, here to prevent unilateral accretions. This is because greater stress is placed on fluid.

the aortic suture line when the clamp is removed, owing to the much higher aortic pressure, and a stent, one-fourth inch, is inserted. The drainage tube with several side holes is inserted. Then the two vascular clamps are now tilted its intrathoracic end is inserted. To avoid each other slightly to provide better exposure of the cut ends of the ductus tunnel made with a curved scissors. The cut ends are inspected to make sure that has been inserted through the medial extent is correctly identified and skin incision low in the eighth intercostal space, no strands of fascia or pleura are present space, bluntly forced subcutaneously where sutures are to be placed. A continuous hemostatic direction and made to fill and clot in the suture needle holes. Three vascular clamps are now placed transversely on the ductus and every effort made to avoid a twisting action on the vessel wall. A second vascular clamp is similarly placed next to the aorta, leaving as much tissue as possible between the two clamps. If less than a 4-mm. segment of the ductus remains, another clamp is placed behind the pulmonary artery clamp, even if it includes more of the wall of the pulmonary artery. The lungs are now fully expanded, removing tension and a normal life span is possible.

The pulmonary side of the ductus is thorax low in the fifth intercostal area is now transected, intact pleura to permit suturing is used a scalpel or very sharp, straight scissors ductus area without undue tension. The cut ends of the ductus is now transected, intact pleura to permit suturing is used a scalpel or very sharp, straight scissors ductus area without undue tension. The cut ends of the ductus is now transected, intact pleura to permit suturing is used a scalpel or very sharp, straight scissors ductus area without undue tension. The cut ends of the ductus is now transected, intact pleura to permit suturing is used a scalpel or very sharp, straight scissors ductus area without undue tension. The cut ends of the ductus is now transected, intact pleura to permit suturing is used a scalpel or very sharp, straight scissors ductus area without undue tension. 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PERSISTENT RIGHT AORTIC ARCH

At various times in embryonic development, there exist six pairs of aortic arches. These undergo complex involutions and transformations as they contribute to the development of the definitive blood vessels. Abnormal development of some of these arches results in the formation of a variety of partial or complete vascular rings surrounding the esophagus and trachea. These vascular rings usually cause significant constriction of the esophagus in dogs rather than tracheal compression as occurs more commonly in human infants (see Comparative Aspects, p. 102).

By far the most frequent type of vascular ring constriction in dogs is that caused by persistence of the right instead of the left fourth aortic arch (Fig. 2). The resultant vascular ring around the esophagus and trachea is composed of the aorta on the right, the ligamentum arteriosum dorsolaterally, the main pulmonary artery on the left, and the base of the heart ventrally. Not infrequently, a retroesophageal left subclavian artery is also present and may contribute to the esophageal constriction. Occasionally, esophageal constriction may be caused by a retroesophageal right subclavian artery arising from a normal left aortic arch. Rarely, both right and left fourth aortic arches persist, forming a double aortic arch with fusion into a single descending aorta just distal to the incarcerated esophagus and trachea.

Persistent right aortic arch (PRAA) occurs most commonly in German shepherd dogs. It also occurs with higher than expected frequency in the Irish setter and Weimaraner breeds.

Although PRAA is a congenital cardiovascular malformation, it is manifested clinically by regurgitation of food due to the esophageal constriction. Aside from the effect on the esophagus, the persistent right aortic arch hemodynamically functions as...
Figure 2. Diagram of the heart, vessels, esophagus and trachea in a dog with persistent right aortic arch viewed from the left side. The esophagus and trachea are incarcerated in a vascular ring consisting of the abnormal aortic arch on the right, the ligamentum arteriosum dorsolaterally, the pulmonary artery on the left and the base of the heart ventrally.

well as a normal aortic arch; therefore, no abnormalities are detected by auscultation, percussion or electrocardiography unless a coexisting hemodynamic lesion is present. The diagnosis of PRAA is made from a history of regurgitation after eating and the demonstration of esophageal dilatation cranial to the heart in barium esophagrams. The esophagus cranial to the constriction at the base of the heart quickly becomes permanently dilated, owing to distention by food and secondary or possibly primary esophageal myoneural junction dysfunction. This was confirmed in one case by an esophageal electromyographic study.

Dogs with PRAA are sometimes in very poor physical condition because of their inability to swallow food and because of aspiration pneumonia. In these cases, an attempt should be made to improve their preoperative status by frequent feedings of baby food in small amounts, in addition to vitamin and iron therapy. Oral treatment for parasites, however, is contraindicated. A serum protein determination should also be made. If surgery is undertaken when the serum protein is under 5 gm. per cent, care must be taken to detect pleural effusion in the immediate postoperative period, owing to low colloid osmotic pressure and pleural capillary damage associated with the thoracotomy.

SURGICAL PRINCIPLES AND TECHNIQUES

The surgical treatment for PRAA is directed toward relief of the vascular ring constriction by dividing the ligamentum arteriosum (Fig. 2). The surgical technique is fundamentally the same as that previously described for dividing a patent ductus arteriosus. Usually, the ligamentum arteriosum is much longer than normal and grossly appears to be a completely fibrous ligament. Occasionally, however, it is still partly patent. Thus, it should always be securely ligated next to the aorta and next to the pulmonary artery before being divided. The pleura should then be dissected from the esophagus 1 to 2 cm. cranial and caudal to the narrowed area, to make certain no fibrous strands remain that will continue to constrict the esophagus.

If a retroesophageal left or right subclavian artery is causing esophageal constriction, either may be divided proximally and distally with ligature-ligatures—and then either may be necessary to reanastomose. To the right of the esophagus in the lateral circulation exists, the steal syndrome has never occur in the dog.

The pleura should not be incised in the area of the ligamentum arteriosum subclavian arteries in order suffering with maximal freedom of the esophagus.

A persistent left cranial turning blood to the right through the coronary sinus is not with persistent right aortic arch is functionally insignificant. A does not open into the left right cranial vena cava is or absent, the persistent right cava may be quite large and surgery for the persistent right cava problem, since it courses across the area of the ligamentum and pulmonary artery. However, dissected free and retracted cranially without too much difficulty.

Proposals have been made to section part of the dilated subclavian arteries in order the placement of sutures in the area of the ligament. At the present time, it appear to be justified for severe 1. With proper management, postoperative regurgitation may take, even though the of the esophagus remains problem, even though the remains intact.

2. It is difficult to know if of the dilated esophagus will volute to achieve a cylinder any constrictions. (a) Dog mature at the time of surgery during may no the time of surgery may dilatation than was present before.

3. Excessive tissue react

*S In occlusive disease of a subgrade flow through the vertebral cerebrovascular anastomoses helps the limb distal to the occlusion when the blood supply to the limb route, cerebral blood flow may be diminished and excessive when the involved limb is been formed the subclavian steal requires angiographic confirmation of cerebral blood flow for diagnosis.
Surgical treatment of congenital cardiovascular diseases—continued

**Surgical Treatment of Congenital Pulmonary Stenosis**

The term pulmonary stenosis encompasses all forms of obstruction to blood flow from the right ventricle to the pulmonary artery. The stenosis may be partial or complete, and can be caused by a variety of factors, including congenital anomalies, fibrous tissue obstruction, or postoperative complications.

**Principles and Techniques**

Surgical treatment for pulmonary stenosis is aimed at relieving the obstruction and improving blood flow to the lungs. The approach to surgery depends on the severity and nature of the stenosis. Common surgical techniques include:

1. **Magnetic resonance imaging (MRI)**: MRI can provide detailed images of the heart and blood vessels, helping to identify the location and extent of the stenosis.
2. **Cardiovascular imaging**: Ultrasound and contrast angiography can be used to visualize blood flow and identify any abnormalities.
3. **Surgical correction**: Depending on the severity of the stenosis, surgical correction may involve the placement of a stent, the creation of a bypass, or the removal of obstructive tissue.

**Pulmonary Stenosis**

Pulmonary stenosis is a common congenital heart defect that affects the blood flow from the right ventricle to the pulmonary artery. The term encompasses all forms of obstruction, whether caused by congenital anomalies, fibrous tissue, or postoperative complications. The goal of surgical treatment is to relieve the obstruction and improve blood flow to the lungs.

**Surgical Options**

- **Stent placement**: A stent can be inserted to create a passageway for blood flow.
- **Bypass surgery**: A bypass can be created to divert blood flow around the stenosis.
- **Tissue excision**: Obstructive tissue can be removed to relieve the stenosis.

**Postoperative Care**

- **Close monitoring**: Postoperative care involves close monitoring of the patient to ensure stable blood flow.
- **Medication management**: Medications may be prescribed to manage symptoms and prevent complications.
- **Follow-up appointments**: Regular follow-up appointments are necessary to assess the effectiveness of the surgical intervention.

**Conclusion**

Surgical treatment of pulmonary stenosis is effective in relieving the obstruction and improving blood flow to the lungs. The approach to surgery depends on the severity and nature of the stenosis, and close monitoring and follow-up care are essential for ensuring long-term success.

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**Note**: This text is a general overview and may not cover all aspects of surgical treatment for pulmonary stenosis. Always consult with a qualified veterinarian for specific recommendations based on your pet's individual needs.
Figure 3. Composite diagram illustrating the most common types of stenoses, occurring singly or in combination, in dogs with obstruction to blood flow from the right ventricle to the pulmonary artery.

SURGICAL TREATMENT OF CONGENITAL CARDIOVASCULAR DISEASES—Continued

the right ventricle to the pulmonary artery. Thus, it may be valvular, immediately subvalvular or infundibular in location (Fig. 3). Pulmonic stenosis occurs about as frequently as patent ductus arteriosus in a hospital clinic population and has been found in a variety of breeds—but more commonly than expected in English bulldogs, Chihuahuas and mixed dogs.

Congenital valvular pulmonic stenosis is the most commonly encountered form and is the easiest to correct surgically, since it is most often caused by fusion of the valve commissures, leaving a small usually centrally located orifice. On rare occasions, however, valvular stenosis may be caused by thickened, immobile valve leaflets without fusion. This is usually associated with hypoplasia of the pulmonic root.

A second type of obstruction has been observed immediately below the valve leaflets. In this form, a complete or partial fibrous ring occurs adjacent to and sometimes involving the base of the pulmonary valve leaflets. Not infrequently this type is associated with thickened, but not fused, valve leaflets and a hypoplastic pulmonic root.

A third site of obstruction is found lower in the right ventricular outflow tract. In this region, hypertrophy of the crista supraventricularis muscle band or of the entire right ventricular outflow tract may cause significant obstruction. In some instances, only a long, narrow channel exists through which blood flows out of the right ventricle. Either type of infundibular hypertrophy may occur as a primary lesion or may be secondary to valvular or immediately subvalvular stenosis.

The clinical features of pulmonic stenosis are described in the preceding section.

The indications for surgery are not always clear, even when cardiac catheterization has been performed. In man, if the gradient is less than 50 mm. Hg (systolic right ventricular pressure is less than 50 mm. higher than systolic pulmonary artery pressure), surgery is not recommended. If the gradient lies between 50 and 100 mm. Hg, surgery is recommended if any clinical signs are evident. If the gradient exceeds 100 mm. Hg, surgery is always recommended.

A sufficient number of dogs with pulmonic stenosis have not been studied under normal environment conditions to establish whether similar or different gradients should be used as indications for or against surgery. It seems reasonable, however, on the basis of cases studied with and without cardiac disability to assume that the indications for surgery in dogs less than one year of age with more than mild signs of disability but in which evidence of more than mild cardiac enlargement is present. Asymptomatic dogs should be examined radiographically for three- to four-month intervals, or more frequently if the dog is over one year of age, whether or not surgery is indicated. It seems unlikely that a dog reaching over one year of age without radiographic evidence of right ventricular enlargement or echocardiographic evidence of right ventricular hypertrophy will ever require surgery.

SURGICAL PRINCIPLES AND RESULTS

Three surgical approaches may be used to relieve the various types of obstruction in pulmonic stenosis. These are similar to those used for heartworms and are accomplished by either a left lateral thoracotomy or a median sternotomy.

The level of obstruction and the type of surgical approach may be determined by pressure studies at cardiac catheterization. It is usually not possible, however, to distinguish between valvular and immediately subvalvular forms of obstruction. Fibrous tissue may exist both above and below, sometimes involving the base of the valve cusps. Angiocardiography is unreliable in distinguishing between the various forms of obstruction.

Although outflow tract obstruction to the pulmonary valve is visible by angiocardiography, it is possible to ascertain whether the primary site of obstruction is either primary or secondary outflow tract obstruction. If the outflow tract obstruction is primary, surgery at the primary site of obstructing fibrous tissue will be required.
cases studied with and without signs of cardiac disability to assume that the values used in man are similar in dogs; as guidelines for recommending surgery. In the absence of cardiac catheterization data, the following indications for surgery are proposed, with the realization that modifications will be made as more experience is gained.

Surgery is indicated regardless of age for any dog with pulmonic stenosis in which signs of cardiac disability are present (dyspnea, weakness, excessive tiring, ascites, edema or hepatomegaly). It is also indicated in dogs less than one year of age without signs of disability but in which radiographic evidence of more than mild cardiac enlargement is present. Asymptomatic young dogs should be examined radiographically at three- to four-month intervals, until they are over one year of age, before deciding whether or not surgery is indicated. It is unlikely that a dog reaching six months of age without radiographic evidence of cardiac enlargement or electrocardiographic evidence of right ventricular hypertrophy will ever require surgery.

**Surgical Principles and Techniques**

Three surgical approaches are applicable to relieve the various types of obstruction in pulmonic stenosis. These approaches are similar to those used for the removal of heartworms and are accomplished through either a left lateral thoracotomy in the fourth intercostal space or a median sternotomy.

The level of obstruction and therefore the surgical approach may be indicated by pressure studies at cardiac catheterization. It is usually not possible, however, to distinguish between valvular stenosis and the immediately subvalvular form, in which the obstructing fibrous tissue is adjacent to and sometimes involving the base of one or more valve cusps. Angiocardiography is also not reliable in distinguishing between these two forms of obstruction.

Although outflow tract narrowing more ventral to the pulmonary valve can be made visible by angiocardiography, it is often not possible to ascertain whether this represents primary or secondary outflow tract hypertrophy. If the outflow tract narrowing is the primary site of obstruction, a median sternotomy approach and a large ventriculotomy are required. However, if the narrowing represents hypertrophy secondary to a valvular stenosis, it need not be resected since studies have shown that sufficient regression of secondary hypertrophy occurs after the primary obstructing lesion has been corrected. In these cases, a simpler transventricular valvulotomy with dilatation will suffice. Angiocardiograms are of additional value in assessing the thickness of the right ventricular wall, since this information is useful for either closed or open ventriculotomy.

In man, greater degrees of poststenotic dilatation of the main pulmonary artery occur more frequently in valvular in contrast to subvalvular pulmonic stenosis. In dogs, however, dilatation of the main pulmonary artery is a less reliable indicator of the level of obstruction.

In some instances, it may be possible to help localize the site of obstruction by direct, one-finger palpation of the right ventricle and pulmonary artery. When preoperative pressure studies are not available or are inconclusive, a small 2-cm.-long thoracotomy can be made in the left fourth intercostal space above the costochondral junction. This can be done just before deciding whether to expose the heart by a median sternotomy or by a large left lateral thoracotomy. Often when right ventricular hypertrophy is present, the outer curvature of this chamber extends in a cranial direction to the root of the pulmonary artery. This is palpable as a transverse shelf at the right ventricular-pulmonary artery junction, and serves to identify the origin of the pulmonary artery without visualization.

If a systolic thrill is palpable only on the wall of the main pulmonary artery, the obstruction is more likely to be valvular stenosis and can be approached through a lateral thoracotomy. If the thrill is palpable on the right ventricular outflow tract as well as on the main pulmonary artery, subvalvular obstruction is more likely present (either immediately subvalvular or a lower infundibular form). In these cases, a median sternotomy is indicated. An additional cause for a systolic thrill in the right ventricular outflow tract is an unsuspected left-to-right shunting interventricular septal defect. However, this can be ruled out after the thorax is entered and the heart is exposed.
If the location of obstruction is uncertain on the basis of cardiac catheterization, angiography or the method of palpation just mentioned, a median sternotomy is the approach of choice. Through this approach either a small or large ventriculotomy can be made, giving access to all forms of pulmonic obstruction. The chief difficulties with this approach are in staying on the midline when dividing the narrow sternum of dogs, and the need for a bone saw.

Valvular Stenosis

When it is fairly certain that a valvular pulmonic stenosis exists, the heart is exposed through a lateral thoracotomy during positive pressure ventilation with caudal displacement of the left apical and cardiac lung lobes.

The stenosis may be relieved by direct vision commissurotomy through a pulmonary arteriotomy during venous inflow occlusion. A longer arteriotomy is required than that needed to remove heartworms; therefore, moderate hypothermia is preferable. As far as technique and time are concerned, this is a more difficult overall procedure; however, it reportedly permits the best anatomic result.

Valvular stenosis can be relieved in a simpler manner by inserting a valvulotome dilator through a small right ventricular outflow tract incision with purse-string control of hemorrhage and no venous occlusion. Special care must be taken to be certain the instrument is inside the lumen before it is turned and directed toward the valve orifice. In an instance in which a dog had a very thick ventricular wall, a myocardial tunnel was made parallel to the outflow tract lumen and contributed to the death of the animal. Either a pulmonary arteriotomy or a closed transventricular valvulotomy can be accomplished through a left fourth intercostal space thoracotomy.

Subvalvular Stenosis

Resection of subvalvular pulmonic stenosis can be accomplished through a median sternotomy and a 3- to 5-cm. incision in the right ventricular outflow tract during venous inflow occlusion (Fig. 4).

After splitting the sternum, a temporary or permanent ligature is tied on the azygous vein. Loose umbilical tape tourniquets are placed on the cranial and caudal veins cavae, and one should also be placed on a persistent left cranial vena cava if this is present. The normally present right cranial vena cava may be small or absent when a persistent left cranial vena cava is present. Failure to detect and occlude this latter vein results in excessive coronary sinus drainage into the right atrium and right ventricle, which obscures vision during the ventriculotomy. The abnormal vena cava can be detected easily by tilting the heart rightward and examining its base. The vein is visible through the mediastinal pleura, coursing in a caudal direction more or less parallel to the left vagus nerve. It enters the pericardium at its cranial reflection on the main pulmonary artery, passes dorsocaudally over the left atrium and opens in the coronary sinus.

The pericardium is incised sufficiently to expose the entire right ventricular outflow tract. Several retention sutures are placed around the edge of the pericardial incision. The weight of hemostats attached to these sutures outside the thorax is sufficient to form a pericardial basket that reduces the motion of the heart.

At this time, the presence or absence of an interventricular septal defect can be ascertained by palpating the right ventricular outflow tract during temporary total venous return. This should be done even if cardiac catheterization has been performed, since a small defect is often missed by nonselective angiocardiography and occasionally by oximetry.

Persistence of a preexisting thrill on the outflow tract for several beats following venous occlusion is evidence of a left-to-right interventricular shunt. Similarly, the development of a thrill when none was present prior to venous occlusion is evidence that a balanced or right-to-left shunt had been present. This becomes left-to-right when the right ventricular pressure drops secondary to arrested venous return. If either type of shunt is thought to be present, an open ventriculotomy is contraindicated and a small purse-string suture controlled ventriculotomy with attempted dilatation of the pulmonic stenosis should be done.

Figure 4. Diagram of the venous tourniquets have been placed on the contemplated incision in the right ventricular outflow tract during venous inflow occlusion (Fig. 4). Just mentioned, a median sternotomy is the approach of choice. Through this approach either a small or large ventriculotomy can be made, giving access to all forms of pulmonic obstruction.
If an existing systolic thrill over the outflow tract of the right ventricle disappears with total venous occlusion, this is considered evidence that the ventricular septum is intact and an immediate subvalvular or infundibular pulmonic stenosis is present. Prior to making a ventriculotomy incision, a sufficient number of loose Cushing type sutures are placed 2 to 3 mm. apart along the contemplated incision line, beginning 1 cm. below the pulmonary valve and extending a distance of 3 to 5 cm. toward the apex. These sutures permit quick closure of the ventriculotomy by simply pulling taut the untied ends (Fig. 4).

Before proceeding with the ventriculotomy, a review of the events that will take place should be made and each person's role fully explained and understood. Of particular importance are the following: proper tightening and later complete release of the vena caval tourniquets, proper functioning of the suction tip and readiness to increase the rate of suction if necessary, proper angle of the suction tip so it does not occlude vision, long forceps and sharp scissors for intracardiac resection of tissue, accurate accounting of time elapsed during venous occlusion, needle holder with needle and suture ready for unexpected sites of hemorrhage, and a well functioning intravenous drip and extra amounts of cross matched blood with arrangements ready for rapid, high-pressure infusion.

When this review is completed, the vena caval tourniquets are tightened, secured with hemostats, and the time is recorded. After three to four heartbeats (to allow ejection of some of the atrial and ventricular blood), an incision is made in the right ventricular outflow tract within the limits of the preplaced row of sutures. This should begin 1 cm. below the pulmonary valve and extend about 3 cm. toward the apex, depending on the size of the heart and the thickness of the ventricle. Lateral retraction of the interatrial reflection on the main conduction nerve. It enters the pericardial cavity, passes dorsocaudally over the diaphragm and opens in the coronary sinus, coursing in a position more or less parallel to the diaphragm and surrounding aortic arch.
cision is accomplished using the untied ends of the preplaced sutures. If the ventricle is quite thick-walled (over 1 cm.), a longer ventriculotomy may be necessary to permit adequate visualization of the lumen. For this reason, it is best to have made preparations for at least a 5-cm. incision with an adequate number of preplaced sutures.

All blood is quickly aspirated from the ventricle, and the suction tip is kept inside the right ventricular apex to remove coronary venous blood. The site of outflow tract narrowing is determined by observation and palpation, and the obstructing tissue is removed. If it involves the base of the pulmonary valve leaflets, then these may also be removed, since in normal dogs excision of the pulmonary valve leaflets produces no demonstrable ill effects. In resecing immediate subvalvular tissue, care should be taken not to perforate the pulmonary artery. If nonresectable tissue appears to continue to obstruct the outflow tract, fracture dilatation may be accomplished with a suitable valve dilator, hemostat or the forceful passage of a finger through the orifice.

After three minutes of venous inflow occlusion at normal body temperature (longer periods with different levels of hypothermia), the suction tip is removed, the ventriculotomy is closed and the vena caval tourniquets are released. Immediate functional closure of the ventriculotomy with minor hemorrhage is accomplished by relaxing the one-fourth-inch umbilical tape suture retractors and pulling taut the preplaced sutures.

The umbilical tape is allowed to fall onto the incision line for added support in temporary hemostasis. It also permits reopening of the ventriculotomy a few minutes later during a repeated period of venous inflow occlusion if this is necessary to complete the intracardiac dissection.

Some hemorrhage occurs through the incision line but is controllable with a gauze sponge, while the preplaced sutures are tied and additional sutures are placed if necessary. The one-fourth-inch umbilical tape can be cut in segments and removed as each preplaced suture is tied.

The suture line can then be reinforced with a deeper layer of interrupted mattress sutures if these are felt necessary. After complete hemostasis is achieved and the heart action appears to be satisfactory, the pericardium is closed, provided this does not impair diastolic filling of the ventricles. Closure of the pericardium should not be watertight, thus avoiding cardiac tamponade should any intrapericardial hemorrhage occur.

Following either a closed valvulotomy or an open subvalvular resection, a palpable thrill is almost always still present on the main pulmonary artery, even though most of the obstruction apparently has been relieved. It is emphasized that, although functional improvement is obtained, surgical treatment does not render the right ventricular outflow tract or pulmonary valve perfectly normal, and turbulent flow usually still exists resulting in a palpable thrill. It has been suggested by some that the pulmonary valve should be dilated until the pulmonary artery thrill is abolished. On the basis of present information, this does not appear to be justifiable and would probably result in rupture of the pulmonary artery root in some instances.

The tourniquet tapes are cut and removed, a chest drainage tube is inserted and the thorax is closed. Postoperative care is the same as described for patent ductus arteriosus (p. 87).

The prognosis for immediate survival and postoperative improvement in dogs with pulmonic stenosis is good when a simple valvulotomy with dilatation can be done to relieve fusion of the pulmonary valve cusps. Attempts to dilate immediate subvalvular fibrous rings by a closed technique in two dogs with intractable congestive heart failure were unsuccessful.

Resection of an immediately subvalvular fibrous rings by a closed technique in two dogs with intractable congestive heart failure were unsuccessful.

The four classic components of Fallot are: pulmonic stenosis, septal defect, overriding aorta, and ventricular hypertrophy. The pulmonic stenosis, which determines the degree of the disorder associated with this condition, decreases blood flow to the lungs and together with the ventricular septal defect accounts for most of the right ventricular pressure elevation. When right ventricular pressure exceeds left ventricular pressure, blood flows from right to left across the septal defect and out the aorta, regardless of the degree of desaturation of the aorta. If right and left ventricular pressures are equal, a balanced shunt exists wherein the degree of the overriding plays a more important role in determining the amount of the systemic blood that enters the systemic circulation.

The resultant cyanosis is a sign of tetralogy of Fallot in dogs. A loud systolic murmur and precordial thrill are present in the pulmonic area (lateral to the costal space below the costochondral junction) when the pulmonic stenosis is extreme. If the obstruction is moderate, a systolic thrill may not be heard. In some instances, evidence of right ventricular hypertrophy is regularly seen. Dyspnea present but may only be marked during exercise. Polythemia secondary to hypoxia may also occur. One million red blood cells per cubic millimeter of blood, a hemoglobin level of 26 gm. per cent, and a hematocrit of 73 per cent are regularly seen. Clubbing of the fingers and toes as reported in man may also be observed in dogs.

Radiographically, dogs with tetralogy of Fallot occasionally have cardiomegaly within normal limits, but usual cardiac enlargement is visible. Prominent pulmonary conus in dorsoventral, lateral, and thoracic projection. The pulmonary arterial pressure is usually normal or increased normally due to poststenotic dilation. The pulmonary conus appears to be constantly increased in size.
TETRALOGY OF FALLOT

The four classic components of tetralogy of Fallot are: pulmonic stenosis, ventricular septal defect, overriding aorta and right ventricular hypertrophy. The first two of these determine the degree of hemodynamic disorder associated with this condition. Pulmonary stenosis decreases blood flow to the lungs and together with the ventricular septal defect accounts for most of the right ventricular pressure elevation and hypertrophy. When right ventricular pressure exceeds left ventricular pressure, unoxygenated blood flows from right to left through the septal defect and out the aorta. This occurs regardless of the degree of dextroposition of the aorta. If right and left ventricular pressures are equal, a balanced or bidirectional shunt exists, wherein the degree of aortic overriding plays a more important role in determining the amount of unoxygenated blood that enters the systemic arteries.

The resultant cyanosis is a cardinal sign of tetralogy of Fallot in dogs. A loud systolic murmur and precordial thrill are present in the pulmonic area (left third intercostal space below the costochondral junction) when the pulmonic stenosis is not extreme. If the obstruction is severe, a murmur may not be heard. In electrocardiograms, evidence of right ventricular hypertrophy is regularly seen. Dyspnea is often present but may only be manifested upon exertion. Polycythemia secondary to chronic hypoxia may also occur. One dog with 11 million red blood cells per cu. mm had a hemoglobin level of 26 gm. per cent and a 73 per cent hematocrit. Clubbing of the extremities as reported in man has not been observed in dogs.

Radiographically, dogs with tetralogy of Fallot occasionally have cardiac silhouettes of extreme size or condition of the patient, the severity of the malformation or the lack of cardiopulmonary bypass facilities, construction of a palliative shunt is indicated rather than just the correction of the pulmonic stenosis by a closed technique. This is because marked concentric hypertrophy of the right ventricle often is present in dogs with tetralogy of Fallot, and only a narrow slit-like lumen may remain in the outflow tract, which cannot be properly managed by a closed procedure.

In cyanotic dogs with pulmonic stenosis and a right-to-left shunting atrial septal defect (or patent foramen ovale), surgical benefit with relief of cyanosis may be obtained from simple closed pulmonic valvulotomy with dilatation, provided this results in a significant decrease in right ventricular pressure. If such is not obtained, then a palliative shunt can also be made.

Palliative Shunts

The surgical procedures described here are
those used in man. Based on a few experimental and clinical studies, they appear to be useful in dogs, with a little modification.

Anastomosis of a subclavian artery to the pulmonary artery (Fig. 5) provides an increase in pulmonary blood flow in cyanotic congenital heart lesions in which the flow of venous blood to the lungs is obstructed and a right-to-left intracardiac shunt exists. In 1945, Blalock and Taussig reported dramatic relief of cyanosis in three “blue” babies by anastomosing either the subclavian artery or innominate artery to a pulmonary artery branch. This form of palliative surgery has subsequently proved beneficial to thousands of children with tetralogy of Fallot, as well as certain other forms of cyanotic congenital heart lesions.

Other palliative shunts that were developed later also are still used in selected cases. In instances in which a subclavian–pulmonary artery anastomosis is not possible, the left pulmonary artery can be anastomosed side to side with the descending aorta (Potts-Smith operation). In tricuspid atresia, an increased pulmonary blood flow can be achieved by anastomosing the cranial vena cava to the right pulmonary artery (Glenn operation). Other palliative shunts have benefited human patients surviving with complete transposition of the great vessels and some form of inadequate spontaneous shunt.

Because both the Blalock-Taussig and Potts-Smith operations are designed to increase pulmonary blood flow by the creation of an artificial patent ductus arteriosus, it is obvious that a spontaneous PDA in cases with tetralogy of Fallot (so-called pentalogy of Fallot) should not be ligated or divided. Anastomosis of a subclavian artery to a pulmonary artery is preferable to side-to-side anastomosis of the left pulmonary artery and descending aorta because less dissection is required, clamping of the aorta is avoided and excessive size of the artificial ductus thus formed is avoided by the self-limiting diameter of the subclavian artery. When a side-to-side left pulmonary artery–aorta anastomosis is made, the size of the shunt can be made too large, thus producing acute congestive heart failure.

Subclavian-pulmonary anastomoses in man are done on the right or left side, depending on the location of the innominate artery as determined by angiocardiography. The procedure is reported to be technically easier when the subclavian artery, arising from the innominate artery, is anastomosed end to side to the corresponding right or left pulmonary artery. Other methods are also reported.

In dogs, no innominate artery exists, the aortic arch extends less caudad, its cranial aspect being the main pulmonary artery and the subclavian artery, which arises from the cranial border of the aorta, is usually long and accessible and is usually long and free from kinking. The operative field is well exposed by a left fourth intercostal space incision with ventral and caudal retraction of the lung lobes. An incision is made in the mediastinal pleura just ventral to the left vagus nerve. While retractor is behind this nerve dorsally, the entire subclavian artery is dissected free as far cranially as possible, where it is divided. Determining the optimum size of anastomosis to the main or left pulmonary artery the pericardium is incised, and the area of the pulmonary artery and visceral pericardium divided. Vessel-to-vessel anastomosis of the subclavian artery is anastomosed to the main pulmonary artery end to end may be angled to preserve the diameter of the arterial orifice for anastomosis. If a persistent left cranial arterial channel is present, it should be dissected and elevated or depressed so it is not compressed by the relocated subclavian artery. The left vagus nerve occurs commonly in human and canine cases of tetralogy of Fallot and was found in a right fourth intercostal space incision made in two dogs with tetralogy of Fallot.

A curved or double angled vascular clamp is applied, and an adequate fold of pulmonary artery is brought forward from the flow of blood under the sternum. One-half the diameter of the main pulmonary artery may be temporarily occluded without harm. If the main pulmonary artery is small for this procedure due to the size of the animal, an end-to-end anastomosis of the left subclavian artery to the left pulmonary artery is performed while the left pulmonary artery is temporarily occluded.

An incision is made in the pericardial area along the cranial border of the aorta for 3 inches. The retractor fold slightly longer than the subclavian artery. Sutures are placed on the medial aspect while the
left pulmonary artery. Other modifications are also reported.

In dogs, no innominate artery exists, and the aortic arch extends less cranially than in man, its caudal aspect being adjacent to the main pulmonary artery. The left subclavian artery, which arises directly from the cranial border of the aorta, is easily accessible and is usually long enough to reach the main pulmonary artery without occlusive kinking.

The operative field is well exposed by a left fourth intercostal space thoracotomy with ventral and caudal reflection of the lung lobes. An incision is made in the cranial mediastinal pleura just ventral to the left vagus nerve. While retracting the vagus nerve dorsally, the entire left subclavian artery is dissected free as far cranial as possible, where it is divided (Fig. 5). After determining the optimum site for anastomosis to the main or left pulmonary artery, the pericardium is incised, and a sufficient area of the pulmonary artery is freed of fat and visceral pericardium to permit proper vessel-to-vessel anastomosis. If the length of the subclavian artery is inadequate, its cut end may be angled to present a larger diameter orifice for anastomosis without stenosis. If a persistent left cranial vena cava is present, it should be dissected free and elevated or depressed so it is not encircled by the relocated subclavian artery. This vein occurs commonly in human patients with tetralogy of Fallot and was found at surgery or post mortem in two dogs and one cat with tetralogy of Fallot.

A curved or double angled partially occluding vascular clamp is applied to exclude an adequate fold of pulmonary artery wall from the flow of blood underneath. Up to one-half the diameter of the main pulmonary artery may be temporarily occluded without harm. If the main pulmonary artery is too small for this procedure due to hypoplasia or the size of the animal, an end-to-side or end-to-end anastomosis of the left subclavian artery to the left pulmonary artery may be performed while the left pulmonary artery is temporarily completely occluded.

An incision is made in the pulmonary artery fold slightly longer than the diameter of the subclavian artery. Suturing is begun at the medial aspect while the vessels are held in approximation to avoid tension and tearing. An everting 1-mm.-wide mattress suture of 5-0 cardiovascular silk with two needles is placed so that knot can be tied on the subclavian artery side. Using the tied ends of the mattress suture, a continuous over-and-over suture pattern with sutures placed 1 mm. apart and everting as much as possible is extended to the dorsal and ventral limits of the anastomosis, where ties are made. The surgeon must carefully place each suture, being certain to include all layers of each vessel wall and avoiding unnecessary trauma, which results in later hemorrhage through needle holes. Hemorrhage from the mediastinal aspect after completion of the anastomosis is virtually impossible to correct with interrupted sutures because of inaccessibility. Therefore, the medial line of anastomosis should be rechecked. Should additional interrupted sutures seem necessary due to a needle laceration or some other reason, these should be placed before continuing the anastomosis laterally. Lateral sutures to complete the anastomosis should be interrupted single or everting mattress types to avoid stenosing the lumen and to permit enlargement with growth. Slight lateral tension on adjacent tied sutures is helpful in elevating the free edges of the vessels to avoid suturing across the lumen when the anastomosis is nearly completed and visualization becomes poor.

After completing the suture line, blood is admitted to fill and clot in the needle holes by slight momentary release of the bulldog clamp on the subclavian artery. When at least three minutes has elapsed, the partially occluding clamp on the pulmonary artery is slowly released. This provides a low-pressure test of the anastomosis. If this is satisfactory, a high-pressure test is made by releasing the bulldog clamp on the subclavian artery. If mild hemorrhage occurs, the clamps are left off and finger pressure is applied up to five minutes before any additional interrupted sutures are placed. Usually, these are not necessary in normal dogs. Cyanotic human patients reportedly may have subnormal clotting ability, and vascular anastomoses may bleed more than expected. This occurred in one small dog with tetralogy of Fallot, in which the left subclavian artery was regarded as too small to...
attempt a Blalock-Taussig procedure and a functioning 3-mm. anastomosis of the left pulmonary to the descending aorta was made. In spite of a usually adequate anatomic technique, persistent hemorrhage occurred, and attempted correction of this proved disastrous when an umbilical tape tourniquet transected the aorta at a previously clamped site.

When all clamps are removed and the anastomosis is dry, evidence of a functioning shunt is obtained by palpating the wall of the pulmonary artery opposite the anastomosis. If satisfactory flow has been achieved, a continuous thrill is palpable. This should not be confused with a systolic thrill in the main pulmonary artery due to the pulmonic stenosis component of tetralogy of Fallot.

When more clinical experience has been obtained with surgery for tetralogy of Fallot in dogs, obviously more reliable recommendations can be made regarding indications for surgery, preference for different procedures, technical problems to be encountered and prognosis for surgical mortality, as well as postoperative improvement and longevity. On the basis of difficulties encountered in performing a left pulmonary artery-descending aorta anastomosis in one case, the fifth left intercostal space appears to provide better exposure than the fourth for this procedure.

COMPARATIVE ASPECTS

It has long been recognized that caution must be used in applying the results of experimental studies in animals to clinical situations in man. Less recognized are the problems of reversing this procedure and transferring carte blanche the information available concerning indications, principles and techniques of cardiovascular surgery in man to somewhat similar conditions in animals. Certain differences in this regard have been observed, and it is likely that additional surgical experience in animals with spontaneous cardiovascular disease will reveal others.

In dogs, the immediate proximity of the aortic arch to the main pulmonary artery and its right branch makes dissection around a patent ductus particularly hazardous. In addition, the external width of a ductus in dogs is usually greater than its length, which rarely exceeds 1 cm., and it is almost always funnel shaped. Also a complication is the routine occurrence of aortic dilatation at the entrance of the ductus, which may assume aneurysmal proportions. On cross section, the dilated part amounts to a double aneurysm composed of the aorta dorsally and rightward and the ductus ventrally and leftward, separated by a distinct intraluminal flap extending a variable distance into the aorta.

Another feature that has impressed this author and has been mentioned by others is the poor ability of the canine aorta to withstand manipulation and to hold sutures. It has been described by a cardiovascular surgeon as "a combination of cheesecake and second-hand Kleenex."

An explanation is also lacking for the apparent absence of aortic coarctation as an entity in dogs. No definite evidence of this lesion has been found in over 200 cases of congenital heart disease, including over 70 cases of patent ductus arteriosus, examined clinically, surgically or at necropsy.

Studies are required to see if there are fundamental characteristics of the canine aorta that account for the apparent absence of coarctation in this species, the apparently greater friability of this vessel and the occurrence of aneurysmal aortic dilatation in dogs with patent ductus arteriosus. Any relationship that these findings might have to the near absence of spontaneous aortic atherosclerosis in dogs should also be investigated.

In young dogs with patent ductus arteriosus and marked cardiac enlargement, postoperative radiograms often show some decrease in heart size but not to the degree usually reported in children. Whether this is because of a simple time-versus-hypertrophy relationship, the frequent postoperative finding of anatomic or functional mitral insufficiency in these dogs or some other factor has not been determined.

A possibly related feature is the occurrence of endocardial splitting of the left atrium in several dogs with patent ductus arteriosus examined at post mortem. Atrial lesions of this type have not been found in other conditions except in valve disease with longstanding left atrial enlargement, splitting is not necessarily evidenced by the occurrence of endothelialized splits in some cases.

The incidence of "ductal" postoperatively in man is low, however, this appears to be the exception in cases studied.

The incidence of "ductal" postoperatively in man is low, however, this appears to be the exception in cases studied. A probable explanation for this occurrence in dogs of funnel that insert obliquely into the aorta.

An explanation is lacking for effects of vascular ring aneurysm and weanling dogs. In infants usually cause tracheal compression and respiratory distress, while in dogs not been observed. It must be the tracheal cartilage in puppies and the tracheal rings are not collapsed. Another difference is greater dilatation of the esophagus.

CARDIAC ARREST

The term cardiac arrest refers to cessation of effective ventricular and atrial contractions. This may be due to either atrial fibrillation. In clinical cardiac arrest is most common in association with anesthesia, hypotension with hypotension with operative pulmonary embolization. Occasionally as a result of external or as a sequel to surgery.

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CARDIAC ARREST

Method of

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The term cardiac arrest refers to an acute cessation of effective ventricular pumping. This may be due to either asystole or ventricular fibrillation. In clinical practice, cardiac arrest is most commonly encountered in association with anesthesia, surgical manipulations, hypotension with shock, postoperative pulmonary embolization and occasionally as a result of electrical shock, overt fear or as a sequel to severe burns.

The prompt recognition of cardiac arrest is of utmost importance because there are only one to two minutes in which to institute effective measures for its correction. The signs are those of total circulatory failure, such as lack of hemorrhage at a surgical wound, absent femoral arterial pulse, disappearance of heart sounds, blanching or cyanosis of mucous membranes and direct evidence of asystole or fibrillation as viewed...

DIAGNOSIS

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on monitors of arterial blood pressure and the electrocardiogram.

The longer the period of ineffective circulation, the more difficult restoration of cardiac and pulmonary function is. The respiratory problems usually result from cerebral hypoxia, particularly in the medullary area, and from lack of circulation through the pulmonary and bronchial circuits. The depression of cardiac function arises from medullary hypoxia and the direct effect of hypoxia on the myocardium. Although there is considerable variation in the time that may elapse before brain damage becomes irreversible, experience with both clinical and experimental cardiac arrest indicates three to four minutes as the usual time.

**PREVENTION**

It is difficult to prevent cardiac arrest with any degree of certainty since its occurrence is largely unpredictable. In certain types of patients, however, the hazard is greater, and these patients should be viewed more critically as potential victims of cardiac arrest. The greatest risk is in animals with disease conditions likely to be accompanied by electrolyte imbalance or toxic states. Patients with chronic renal, hepatic or gastrointestinal disease, uremia, metabolic acidosis and burns are in this category. Animals with heart disease also should be handled with increased caution. However, this has been overemphasized to a great extent, for the incidence of cardiac arrest in this group is very low except when incipient or frank congestive heart failure exists. Prolonged abdominal surgery increases the risk of arrest, especially if much traction on the abdominal wall and viscera is required. Thoracic surgery poses no additional risk of cardiac arrest provided ventilation is adequate and care is exercised in retracting heart and lungs so as not to interfere with venous return to the right and left atria. Cardiac surgery, especially open cardiotomy, poses unique problems that normally are not encountered.

Proper preanesthetic medication is of value in preventing cardiac arrest during induction or during the anesthetic period itself. This is especially true in the high-risk group or in very apprehensive patients. Promethazine (Phenergan) or promethazine and promazine combined are satisfactory agents for this purpose and do not depress the medullary centers as greatly as opiates, although opiates are also used successfully. Atropine sulfate (0.05 mg./lb. of body weight) is often given to prevent vagotonic reflexes, and it is probably more useful in abdominal and orthopedic procedures than in thoracic procedures, with the exception of lobectomy or pneumonectomy, in which clamping of a bronchus sometimes produces notable reflex showing of the heart, or occasionally cardiac arrest. The irritability of the heart during manipulations is not altered by atropine, and in few animals it actually seems increased. Once anesthesia is achieved, an endotracheal tube should be inserted, and in the poor risk group it is advisable to administer oxygen and artificial respiration since ventilation during anesthesia is frequently inadequate. The use of inhalant anesthetic agents is recommended because of their rapid elimination when administration ceases. One should make certain that an intravenous route for the administration of fluids and medication is available.

Some warning of impending cardiac arrest always appears, although it may be only several seconds before arrest occurs. Monitors of the electrocardiogram and arterial blood pressure provide the necessary information in almost every case. The electrocardiogram usually shows arrhythmias that precede ventricular fibrillation, and it often shows profound cardiac slowing prior to asystole. Arterial blood pressure usually declines precipitously or becomes zero when cardiac arrest is imminent or occurs. The electrocardiogram does not provide reliable evidence of asystole because normal electrical activity may take place for several seconds or minutes after mechanical cardiac action has stopped. When any of these warning signs occur, all manipulations and administration of anesthetic agents should be stopped until normal cardiac activity resumes. Further manipulations should then be carried out carefully and signs of recurring difficulty carefully watched for.

Procaine derivatives have been employed to prevent or abolish arrhythmias, but they are not recommended because of their myo-