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Appendix 6.9 of James W. Buchanan's dissertation Chronic Valve Disease and Left Atrial Splitting in the Dog

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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).

Figure 1. Diagram of the heart and great vessels of a dog with patent ductus arteriosus (P.D.A.) viewed from the left side. Characteristically, the aorta is dilated where the ductus enters obliquely in line with the long axis of the right ventricular outflow tract (R.V.). Right auricle, R.A.; left auricle, L.A.; left ventricle, L.V.; left subclavian artery, L.S.A.; and brachiocephalic trunk, B.T.
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 is 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 annular dilatation. This is usually not detected until after the PDA has been corrected. In some instances, the heart size later decreases noticeably 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 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 conges-

tive 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 two cases that failed 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 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 occluding 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 that they do not have a tendency to slide together. In these cases, an additional transfixed suture-ligation should be placed between the two circum-

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Surgical Treatment of Congenital Cardiovascular Diseases—Continued

In most dogs, the width of the PDA is greater than the external length and the ductus is wedge shaped, so that ligatures tend to slide to the narrowest point—usually adjacent to the pulmonary artery. This type of ductus does not permit two separate ligatures. It is inadvisable to depend on one ligature in these cases, because instances of later recurrence of flow through the ductus have been observed. It is, therefore, advisable in most, if not all, instances of PDA in dogs to divide and oversew the ductus between two occluding vascular clamps.

The thorax ordinarily is entered through a left fourth intercostal space incision, extending dorsally to the edge of the longissimus dorsi muscle and ventrally just below the costochondral junction. In dogs with marked cardiac and pulmonary artery enlargement, the fifth interstice provides better exposure. After initiation of positive pressure ventilation, the fourth and fifth ribs are spread apart as far as possible, and the left apical cardiac and lung lobes are reflected caudally and cranially ventral to the pulmonary artery, main pulmonary artery, and cranial to the ductus itself. The pericardium, an incision is made in the mediastinal pleura, slightly above the level of the ductus after branching off the left vagus nerve. An incision in the mediastinal pleura between the aorta and pulmonary artery. In these cases, extreme caution is required in dissecting the craniomedial 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.

In most dogs, however, the cranial part of the funnel-shaped ductus is less than 1 cm. in length and may only constitute a 1- to 2-mm-long curve as it connects the aorta and pulmonary artery. In these types, it is usually necessary to dissect both intrapericardially and extrapericardially to get around the ductus. A good plane for intrapericardial dissection of the ductus can usually be entered after opening the pericardial sac near its dorsal reflection on the pulmonary artery at the entrance of the ductus just ventral to the left vagus nerve. With lateral traction on the dorsal cut edge of the pericardium, an incision is made into the dorsal pericardial reflection with scissors, and a communication with the more dorsal dissection plane is established. At this point, a loose retraction ligature is placed around the vagus nerve to keep it out of the way. Dissection is then continued by incising in both cranial and caudal directions along the...
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 so severed ends of the ductus to permit it may be seen if any blood is getting interrubbing of sutures on opposing walls of the supposedly occluded segment. If so, then to aid in establishing firm blood clots, the nonoccluding clamp (determined by the needle holes. The opening in the color of the blood) is closed another notch dium is now closed, and if there is.

The ductus is now completely transected, intact pleura to permit suturing is done using a scalpel or very sharp, straight scissors ductus area without undue tension, care should be taken to avoid serrating, is done. However, if this causes a cut edge. If less than a desirable amount of vascular or nerve tissue, of ductus tissue is present between the oc-ductus area is left exposed. An additional suturing the cut edges, the division is made off-cen-made in the ventral mediastinum so that more is left on the aortic side, the heart to prevent unilateral access. This is because greater stress is placed on fluid through the aortic suture line, when the clamp is.

The lung is now repositioned, owing to the much higher aortic pressure, and a stiff, one-fourth inch drainage tube with several side holes is placed behind the pulmonary artery side of the ductus. The initial clamp on the pulmonary artery is arrested by this time. Usually, it is necessary before closing the skin incision to make sure that has been inserted through the medial extent is correctly identified and skin incision low in the eighth thoracic region, where no strands of fascia or pleura are present, blast and then approximated with 2-0 medium chromic gut. The clamps are still exposed and not attached to the heart to prevent unilateral access. The cut ends are inspected to make sure that has been inserted through the ductus area without undue tension, bluntly forced subcutaneously where sutures are to be placed.

The pulmonary side of the ductus is su-thorax low in the fifth intercostal space, being transected with 5-0 Atraloc silk in a continuous mattress suture. The cut ends are inspected to make sure the cut edges, the division is made off-center so that more is left on the aortic side, the heart to prevent unilateral access. The prognosis for successful surgical treatment is guarded in some cases; however, the ductus is divided.

The suture method is repeated on the by suction with a syringe while the aortic side of the ductus. Usually, it is nec-less temporarily released. The tube is removed, owing to the much higher aortic pressure, and a stiff, one-fourth inch drainage tube with several side holes is placed behind the pulmonary artery side of the ductus. The initial clamp on the pulmonary artery is arrested by this time. Usually, it is necessary before closing the skin incision to make sure that has been inserted through the ductus area without undue tension, bluntly forced subcutaneously where sutures are to be placed.

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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
SURGICAL TREATMENT OF CONGENITAL CARDIOVASCULAR DISEASES—Continued

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 ligature-ligated and then (if necessary to reanastomose a lateral circulation exists, a steal syndrome* has never occurred in the dog.

The pleura should not be sutured to the area of the ligamentum arteriosum subclavian arteries in order to prevent maximal freedom of the esophagus.

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

Proposals have been made for section of part of the dilation of the placement of sutures. At the present time, it appear to be justified for several:

1. With proper management, postoperative regurgitation, problem, even though the problem, even though the esophagus remains

2. It is difficult to know of the dilated esophagus volvulus and to achieve a cylinder with any constrictions. (a) Dogs mature at the time of surgery during may not be tolerated than was present before.

3. Excessive tissue react-

*In occlusive disease of a sub in occlusive disease of a sub grade flow through the vertebral cerebrovascular anastomoses help to the limb distal to the occlusion when the blood supply to the limb route, cerebral blood flow may be compromised and signs of cerebral hypoxia may be seen when the involved limb is occluded by the subclavian stea- quires angiographic confirmation of the subclavian arterial blood flow for diagnosis.
Surgical Treatment of Congenital Cardiovascular Diseases—Continued

In a dog with persistent right aortic arch, a vascular ring consisting of the aorta, the pulmonary artery on the left, and the ligamentum arteriosum is described in a vascular ring consisting of the aorta, the pulmonary artery on the left, and the ligamentum arteriosum. The thoracic aorta and subclavian arteries are normally longer than normal and grossly abnormal or absent. The persistent left cranial vena cava may be quite large and interfere with surgical ligation of the aorta and subclavian arteries in order to avoid interfering with maximal freedom of movement of the esophagus.

A persistent left cranial vena cava returning blood to the right atrium by way of the coronary sinus is not unusual in dogs with persistent right aortic arch. This vein is functionally insignificant as long as it does not open into the left atrium. If the right cranial vena cava is abnormally small or absent, the persistent left cranial vena cava may be quite large and interfere with surgery for the persistent right aortic arch problem, since it courses directly over the area of the ligamentum arteriosum and the pulmonary artery. However, it can be dissected free and retracted dorsally or ventrally without too much difficulty.

Proposals have been made advising resection of part of the dilated esophagus or the placement of sutures to invert a portion. At the present time, this does not appear to be justified for several reasons.

1. With proper management of food intake, postoperative regurgitation is not a problem, even though the cranial portion of the esophagus remains dilated.

2. It is difficult to know just how much of the dilated esophagus to resect or invert to achieve a cylindrical tube without any constrictions. (a) Dogs are usually not mature at the time of surgery. (b) Anesthesia during surgery may cause more dilatation than was present before anesthesia.

3. Excessive tissue reaction and scarring following esophageal surgery may cause later constriction.

4. Peristaltic activity in the esophagus usually does not return to normal and may be compromised even further by esophageal surgery.

5. There is increased chance for postoperative complications, such as esophageal pleural fistulas and infection.

The thorax is closed in routine fashion and a drainage tube is left in place as long as necessary to make certain pleural effusion does not develop.

Immediate postoperative care is the same as that described for patent ductus arteriosus, with the additional problem of food management. Small amounts of soft baby food are given several times daily for the first two weeks in addition to liquid oral vitamins. After this time, food may be given in various forms to determine the amount, frequency, and kind of food that the animal manages best. In one case in which vomiting and poor physical condition continued to be a problem for months in spite of the usual regimen of small amounts of soft food several times daily, it was found by accident that the dog did very well on dry kibbles ad libitum placed at head height. This dog previously bolted down soft foods, which it would then regurgitate; however, it could swallow the dry form, which it had to chew into small pieces.

The prognosis for dogs with persistent right aortic arch is favorable if they are not too emaciated to permit surgery. The surgical relief of esophageal constriction is relatively simple and carries a low mortality rate. The chief problem in these cases is the need for continued management of food intake, for esophageal dilatation has persisted postoperatively in all dogs studied.

In one case just mentioned, marked esophageal dilatation and abnormal peristaltic activity were still present on the basis of fluoroscopic studies 15 months after surgery. The dog was in good physical condition, however, and had not regurgitated food for months.

PULMONIC STENOSIS

The term pulmonic stenosis encompasses all forms of obstruction to blood flow from
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.

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. 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 indications used in man are similar in dogs for recommending surgery.

Surgery is indicated regardless of the existence of cardiac catheterization studies providing indications for surgery. The indications for surgery are based on the realization that right ventricular outflow tract hypertrophy is present. Asymptomatically dogs examined radiographically are over one year of age, whether or not surgery is recommended depends on whether or not surgery is indicated in dogs less than one year of age without radiographic evidence of right ventricular hypertrophy or evidence of right ventricular outflow obstruction.

Surgical Principles and Approaches

Three surgical approaches may be employed to relieve the various types of stenosis in pulmonic stenosis. These are similar to those used for heartworms and are accomplished via either a left lateral thoracotomy or an intercostal space approach. The level of obstruction and the surgical approach may be determined by pressure studies at cardiac catheterization. It is usually not possible to distinguish between valvular and immediately subvalvular forms of obstruction. Fibrous tissue is sometimes involving the base of the valve cusps. Angiocardiography is reliable in distinguishing between primary or secondary outflow tract obstruction.

Although outflow tract hypertrophy is visible by angiocardiography, it is possible to ascertain whether the primary site of obstruction is left, right or both ventricles. If the outflow tract is dilated at the primary site of obstruction, an atrial septal defect approach and a large
ventricular outflow tract. In this instance, hypertrophy of the crista supraventricular band or of the entire right ventricular wall occurs. Either subvalvular or immediately subvalvular obstruction 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 with flow from the right ventricle to the pulmonary artery.

**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 the 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, angiocardiography 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 pulmonic 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 azygos vein. Loose umbilical tape tourniquets are placed on the cranial and caudal venae 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.

Tourniquete caudal vena cava is intact and an immediate infundibular pulmonic stenosis.

Prior to making a ventriculotomy, a sufficient number of loose sutures are placed 2 to 3 cm below the pulmonary valve, a distance of 3 to 5 cm from the right ventricular apex. These sutures permit the ventriculotomy by simulating the untied ends (Fig. 4).

Before proceeding with the ventriculotomy, a review of the event place should be made and carefully explained and understood. The importance are the following: tightening and later complete venous caval tourniquets, protection of the suction tip and ready
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 incision is contraindicated and a spring suture controlled venous return attempted dilatation of the tourniquets should be done.
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, these may also be removed, since in normal dogs excision of the pulmonary valve leaflets produces no demonstrable ill effects. In resecting 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 respiratory effort is abolished. On the basis of present information, this does not appear to be justified 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 resultant cyanosis is a feature of tetralogy of Fallot in dogs with a pulmonic stenosis and a ventricular septal defect, overriding aorta, increased pulmonary blood flow, and the presence of a large patent ductus arteriosus. The resultant cyanosis is also frequently present in man. Cyanosis may be so marked as to render the animal unemployable as a circulatory test animal. Cyanosis is also frequently present in dogs with intractable congestive heart failure. Cyanosis is frequently observed in dogs with pulmonic stenosis. Cyanosis is also frequently present in dogs with intractable congestive heart failure.

The four classic components of tetralogy of Fallot are: pulmonic stenosis, ventricular septal defect, overriding aorta, and right ventricular hypertrophy. The presence of these four components is diagnostic of tetralogy of Fallot. Pulmonic stenosis decreases blood flow to the lungs and together with the ventricular septal defect accounts for most of the pulmonary pressure elevation. When right ventricular pressure exceeds left ventricular pressure, blood flows from right to left across the ventricular septal defect and out the aorta. If the right and left ventricular pressures are equal, a balanced shunt exists, wherein the degree of right-to-left shunting is determined by the amount of blood that enters the systemic circulation.

The resultant cyanosis is a feature of tetralogy of Fallot in dogs. The cyanosis is present in dogs with pulmonic stenosis and a ventricular septal defect, overriding aorta, increased pulmonary blood flow, and the presence of a large patent ductus arteriosus. The resultant cyanosis is also frequently present in man. Cyanosis may be so marked as to render the animal unemployable as a circulatory test animal. Cyanosis is also frequently observed in dogs with pulmonic stenosis. Cyanosis is also frequently present in dogs with intractable congestive heart failure.
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. Pulmonic 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, unoxgenated 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 unoxgenated 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 within normal limits, but usually right heart enlargement is visible. Prominence of the pulmonary conus in dorsoventral radiograms due to poststenotic dilatation of the main pulmonary artery is not usually seen. Instead, the pulmonary conus segment may be concave owing to hypoplasia of the pulmonary arteries and cranial displacement of the adjacent aortic arch segment.

All the clinical signs observed in dogs with tetralogy of Fallot may also be seen in dogs with pulmonic stenosis and with some form of atrial septal defect in which elevated right atrial pressure exists. This causes shunting of unoxgenated blood into the left atrium, and peripheral cyanosis. In these dogs, however, prominence of the pulmonary conus due to poststenotic dilatation of the main pulmonary artery usually can be seen in dorsoventral radiograms.

A differential diagnosis can be made by cardiac catheterization, in which the level of right-to-left shunt can be determined either by observing the course of the catheter, oximetry or selective angiocardiography.

SURGICAL PRINCIPLES AND TECHNIQUES

Definitive corrective surgery for tetralogy of Fallot consists of relieving the pulmonic stenosis by valvulotomy or subvalvular resection, and closing the ventricular septal defect by simple suturing or patch grafting during cardiopulmonary bypass. When corrective surgery is not possible due to the 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 angiography. 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 forms are also reported.

In dogs, no innominate artery is present. The aortic arch extends less caudally in the dog than in man, its caudad aspect being less accessible and is usually long and kinked.

The operative field is well exposed by extending the left fourth intercostal space posteriorly to the vertebral column. The left fourth intercostal space contains the lung lobes. An incision is made in the mediastinal pleura just lateral to the left vagus nerve. While retractor blades elevate the left vagus nerve dorsally, the entire innominate artery is dissected free as far as possible, where it is divided. Anastomosis to the main or left pulmonary artery is made after the pericardium is incised, and the whole area of the pulmonary arteries and visceral pericardium is cleared by vessel-to-vessel anastomosis. Anastomosis of the subclavian artery is also done end to end may be angled to preserve the arterial diameter orifice for anastomosis. If a persistent left cranial mediastinal pleura just lateral to the left vagus nerve is present, it should be dissected, elevated or depressed so it is not in the way of the relocated subclavian artery. Anastomosis occurs commonly in human and can be done during surgery or post mortem in two dogs with tetralogy of Fallot.

A curved or double angled vascular clamp is applied to the aorta. An adequate fold of pulmonary artery is exposed from the flow of blood under the aortic arch. The size of the vascular anastomosis is one-half the diameter of the main pulmonary artery, which is usually the size of the vessel to be anastomosed. The size of the vessel to be anastomosed is determined by the size of the animal, an end-to-end anastomosis of the aorta to the main pulmonary artery is performed while the left pulmonary artery is temporarily occluded.

An incision is made in the pulmonary artery fold slightly longer than the diameter of the subclavian artery. Sutures are placed through the medial aspect while the arterial incision is made.

**Figure 5.** Diagram of the Blalock-Taussig operation in dogs. The left subclavian artery (L.S.A.), before being divided and reflected caudally, is situated dorsal and parallel to the brachiocephalic trunk (B.T.). End-to-side anastomosis of the left subclavian artery to the main pulmonary artery (M.P.A.) provides increased pulmonary blood flow in dogs with tetralogy of Fallot.
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 to the left vagus nerve. After 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 detennining 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 adequate, 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 completely 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 evertine 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 evertting 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 medial 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 evertting 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 apparently absent 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 athero- sclerosis 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 and left atrial enlargement splitting is not necessarily evidenced by the occurrence of endothelialized splits in some.

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 anomaly and weaning dogs. In infants, usually cause tracheal collapse and respiratory distress, while in the dog, it is not been observed. It must be that the tracheal cartilage in puppy and the tracheal rings are collapsed. Another difference is the greater dilatation of the esophagus.

The term cardiac arrest refers to cessation of effective ventricular action. This may be due to either atrial or ventricular fibrillation. In clinical medicine, cardiac arrest is most common in association with anesthesia, manipulations, hypotension with operative pulmonary embolectomy, occasionally as a result of overt fear or as a sequel to a...
other conditions except in chronic mitral valve disease with longstanding insufficiency and left atrial enlargement. Endocardial splitting is not necessarily a fatal event, as evidenced by the occurrence of well healed endothelialized splits in some dogs.

The incidence of "ductus diverticulum" postoperatively in man is low. In dogs, however, this appears to be the rule rather than the exception in cases studied thus far. A probable explanation for this is the frequent occurrence in dogs of funnel shaped PDA's that insert obliquely into the descending aorta.

An explanation is lacking for the different effects of vascular ring anomalies in infants and weanling dogs. In infants, these anomalies usually cause tracheal compression with respiratory distress, while in dogs this has not been observed. It must be suspected that the tracheal cartilage in puppies is stronger, and the tracheal rings are less easily collapsed. Another difference is that much greater dilatation of the esophagus seems to occur in dogs. One possible explanation for this is that dogs begin to eat more solid foods at a much earlier age. Another factor may be that the long axis of the esophagus in dogs always parallels the ground. This position increases the gravity stress on the esophageal wall when ingesta is present, while decreasing the advantage of gravity in aiding the passage of food to the stomach.

The higher incidence of certain congenital heart diseases in specific breeds of dogs provides a unique opportunity for selective breeding and the study of genetic factors in the etiology of various congenital heart lesions. Most promising in this regard is the higher incidence of patent ductus arteriosus in poodles, persistant right aortic arch in German shepherds, Weimaraners, and Irish setters, pulmonic stenosis in English bulldogs and Chihuahuas, and subaortic stenosis in boxers and German shepherds.

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 a 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-