mesentery which respond to engorgement of the neighboring tissue by raising the systemic blood pressure (*i.e.* arterial vasoconstriction). They also showed that these receptors would respond in the same manner to increased pressure in the abdominal vena cava. It is possible that these receptors are the initiators of the observed reflex.

Should this be true, then it is understandable why the expiration may not produce diminution of pulse and finger volume if not forced. It is likely that a critical intrathoracic pressure must be reached before the abdominal venous pressure is raised enough to stimulate the receptors. In that event, a deep expiration that does not do this will not elicit the reflex.

*Summary.* 1. Twelve records of the effect of a deep, forced expiration on finger pulse volume were obtained from six subjects. In four of these records a ballistocardiogram was recorded simultaneously. In 5 other tracings total finger volume changes were recorded.

2. In all cases the pulse volume and total finger volume (when recorded) decreased immediately following the deep forced expiration. Average duration of diminution was 16 seconds for pulse volume and 24 seconds for total finger volume.

3. The absence of a simultaneous decrease in corresponding ballistocardiogram beats suggests that the observed finger changes were due to reflex arteriolar constriction, possibly initiated by abdominal presso-receptors in the great veins or mesentery.

Appreciation is expressed to Prof. William S. McCann for his advice and criticism in the review of the manuscript, and to Prof. W. O. Fenn for his helpful suggestions during the course of the work.

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## Location of Communications between Cognate Bed of Descending Ramus of Left Coronary and Adjacent Collateral Vascular Beds.*

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More than 40 years ago, Hirsch and Spalteholz[1] noticed that after ligation of a coronary artery, the area of infarction was much smaller than the myocardium apparently supplied by that artery (cognate bed). Two years later Miller and Mathews[2] made the same observations, and suggested that this was due to the presence of fine epicardial arterial anastomoses. Since that time, numerous additional reports have appeared describing the variability in the occurrence of and in the size of infarcts produced by coronary artery ligation.[3-8] Sutton and Davis[9] suggested tying the superficial anastomotic vessels in order to get a large infarct.

No actual studies, however, appear to have been done in order to demonstrate whether or not the blood supplied by the superficial

* Supported by a grant from the Life Insurance Medical Research Fund.

1 Hirsch, C., and Spalteholz, W., quoted by L. Gross, 1921, Hoeber, N. Y.

2 Miller, J. L., and Mathews, S. A., *Arch. Int. Med.*, 1909, **3**, 476.

3 Smith, F. M., *Arch. Int. Med.*, 1918, **22**, 8.

4 Mendlowitz, M., Schauer, G., and Gross, L., *Am. Heart J.*, 1937, **13**, 663.

5 Karsner, H. T., and Dwyer, J. F., *J. Med. Research*, 1916, **34**, 21.

6 Smith, F. M., *Am. J. Med. Sc.*, 1918, **156**, 706.

7 Gold, H., Travell, J., and Modell, W., *Am. Heart J.*, 1937, **14**, 284.

8 O'Neill, J. F., and Thomas, W. C., Personal communications, 1947.

9 Sutton, D. C., and Davis, M. D., *Arch. Int. Med.*, 1931, **48**, 1118.

vessels is the cause of the variability in the resulting infarctions. It was reasoned that if the principal collateral supply is through the superficial vessels, then if an artery were ligated and all superficial anastomotic branches were interrupted, an infarct of fairly predictable size should result. On the other hand, if a consistent infarct was not obtained, then the collateral blood supply must come by way of deep communicating vessels. This was tested by the following procedure:

*Method.* Mongrel dogs of both sexes and weighing 5 to 16 kg were anesthetized with intravenous sodium pentobarbital† (40 mg/kg), artificial respiration was administered and aseptic thoracotomies performed by subperiosteal rib resection.

Immediately before operation the dogs were given 10,000 units of penicillin. Postoperatively they received a total of 90,000 units in divided doses 3 times a day for 3 days. In the last 24 thoracotomies penicillin in emulsified oil (Pendil‡) was used to maintain the penicillin level through the 2 first postoperative nights with water soluble penicillin being given in the day. This materially reduced the number of injections required and was found to be much more convenient. In these series totalling 52 thoracotomies treated with penicillin there was not a single death from infection nor did any show more infection than a drop of pus around an unremoved skin suture. This is in marked contrast to Miller and Mathews'[2] results of a death rate of 60% due to sepsis.

Three groups of procedures were carried out upon the hearts.

*Group A.* In 23 dogs the anterior descending arteries and the accompanying veins were ligated 1 to 2 cm below the bifurcation of the left coronary arteries. This is the region where there is usually a relatively large branch going to the left ventricular myocardium. In 11 of these dogs the main vessel and this large branch were individually tied: in 9 dogs the ligature was above the first large branch; in 3 dogs the ligature was below the first main branch which was left intact (see diagram). The areas of distribution of the arteries were exposed and the edges touched with a hot iron cautery producing burns light brown in color and about 3-5 mm in width. Similar burns on excised and living hearts showed that burns of this type extend into the myocardium less than a millimeter. The chests were closed in layers (Fig. 1).

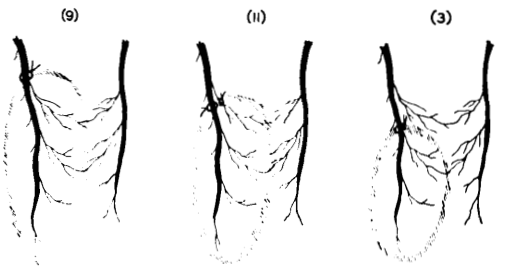


FIG. 1.
Diagram of the site of ligation of the descending ramus of the left coronary artery with the site of the superficial burn indicated by crosshatching. The figures at the top of the diagram indicate the number of animals receiving each type of ligation plus cautery. All of the above types are included in Group A.

*Group B.* In 8 animals a ligation was done as described for the first 20 of Group A but cauterization was omitted.

*Group C.* In 4 animals cauterization as described was done without ligation.

All animals were sacrificed 8 to 14 days after operation.

*Results.* Table I. Six animals died within 24 hours after the ligation-cauterization procedure. The cause of death was probably ventricular fibrillation since no other cause could be found at autopsy. These animals were dropped from the series because their early deaths did not allow enough time for the development of gross evidence of infarction. Recently preoperative and postoperative intravenous procaine, 10 mg/kg at each administration, was added to the procedure. Of 6 dogs so treated, one died of fibrillation while the chest was still open. The remainder survived until sacrificed. The latter death casts some doubt on the efficacy of procaine in preventing the fibrillation which occasionally accompanies this procedure.

† Supplied by Premo Pharmaceutical Labs, Inc., New York City, N. Y.

‡ Supplied by Endo Products, Inc., Richmond Hill, N. Y.

TABLE I.
Results in Animals Sacrificed.

| Groups | Gross infarction | No gross infarction | No. in group |
|---|---|---|---|
| Group A | 17 | 0 | 17 |
| " B | 6 | 2 | 8 |
| " C | 0 | 4 | 4 |
| Total in Groups A and B | 23 | 2 | 25 |

The difference in occurrence of infarction was significant below the 5% level. In this case:

$$X^2 = \frac{(ad - bc)^2 N}{(a+b)(c+d)(a+c)(b+d)} = 4.619^{10}$$

The results on the remaining animals were as follows: *Group A.* Cautery and Ligation. All 17 of the hearts of the animals surviving the first 24 hours showed definite gross infarctions involving the full thickness of the myocardium and extending to the burned areas.

*Group B.* Ligation. Two of these showed extensive infarctions approximating those seen in Group A; 4 showed infarctions smaller than area apparently supplied by the artery; 2 showed no gross evidence of infarction. All areas of necrosis stopped abruptly at the right edge of the anterior vessels, *i.e.,* the necrosis did not involve the right ventricular myocardium. These were different than the ligated cauterized infarctions where the necrosis extended to the cauterized area on the right ventricle. None of the infarctions in either series involved the interventricular septum.

*Group C.* The only lesions found were adhesions between the burned area and the pericardium. None showed gross infarctions.

*Discussion.* The $X^2$ value of $4.619^{10}$ comparing incidence of infarction in Groups A and B, shows that the probability of chance occurrence lies well below the 5% level. Therefore, we consider this difference to be probably significant.

Lowe[11] and Lowe and Wartman[12] have shown how branches from the epicardial vessels go to their respective layers of the myocardium. It appears possible from our studies that these small arteries may be end arteries, in the strict sense, and have only capillary and small arteriolar anastomoses with the other arteries supplying the same layers. The present study suggests that the deep small anastomosing vessels cannot prevent necrosis of a very large portion of myocardium when its epicardial supplying arteries are interrupted.

*Summary and conclusion.* The frequent failure to obtain infarcts and the variability in the size of the infarcts which follow ligation of a coronary artery in the dog suggest the presence of prominent anastomotic communications with collateral vascular beds. To determine whether these communications were in deep or superficial vessels, the consistency of infarction with simple coronary ligation was compared with the infarction which resulted from coronary ligation plus occlusion of the superficial vessels by cauterization about the cognate bed of the ligated vessels. Since the latter procedure produced infarctions of predictable size in all of the 17 dogs so treated, which survived for 24 hours or longer, it is concluded that the principal anastomotic communications with collateral vessels lie in the epicardium.

It is suggested that ligation plus superficial cauterization may be of definite value in providing a fairly well standardized procedure for the experimental study of myocardial infarction.

10 Chambers, E. G., Cambridge Univ. Press, 1940, Cambridge, England.

11 Lowe, T. E., *Am. Heart J.*, 1941, **21**, 326.

12 Lowe, T. E., and Wartman, W. B., *Brit. Heart J.*, 1944, **6**, 115.