curves for autolysis and for lysis. The protein must, therefore, be hydrolyzed immediately upon disintegration of the cell.

In the case of *B. coli* (Fig. 4) or *B. megatherium* (Fig. 5), however, some protein does appear in solution as the cells disintegrate, since the decrease in water-insoluble protein during lysis is faster than the decrease in total protein. The total protein also decreases before lysis is complete and at the end of the process the solution is practically protein-free.

The coli and megatherium could not be made to autolyze.

According to these results there is no general relationship between lysis and protein-hydroysis with different basterial suspensions. No protein appears in solution during lysis or autolysis of staphylococcus while some does appear during lysis of *B. coli* or *B. megatherium.*

The writer is indebted to Dr. A. P. Krueger for the *Staphylococcus aureus* cultures and phage and to Professor André Gratia for the *B. coli* and *B. megatherium* cultures and their accompanying phages.

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Immediate Effects of Coronary Sinus Ligation on Dynamics of Coronary Circulation.

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It has been suggested by Gross,[1] Robertson,[2] and Ungerleider[3] that ligation of the coronary sinus or coronary veins might materially aid hearts whose normal blood supply had been partially removed. Fortunately, such a concept can be tested physiologically and hence an experimental study was made of the possible practical value of such a procedure.

In order to obtain a better understanding of what happens dynamically in the coronary vascular bed following ligation of the coronary venous system, the following have been studied in 28 acute experiments on dogs and largely by optical methods: (1) the coronary

[1] Gross, L., Blum, L., and Silverman, *J. Exp. Med.*, 1937, **65**, 91.

[2] Robertson, H. F., *Am. Heart J.*, 1935, **10**, 533.

[3] Ungerleider, H., Kerkhof, A., and Fahr, G., PROC. SOC. EXP. BIOL. AND MED., 1938, **37**, 703.

inflow, (2) the peripheral coronary pressure, (3) intravenous pressure, (4) the retrograde blood flow in the coronary arteries, together with oxygen determinations, (5) the ability of the myocardium to maintain contractions after its normal blood supply has been removed. The results which have been obtained follow.

After coronary sinus ligation the reduction in coronary inflow (by the method of differential pressure curves) is considerable in the left coronary, but not in the right.

The contour and time relations of the peripheral coronary pressure are similar to those obtained under normal conditions[4] but both the diastolic and systolic resistances are greatly elevated in the left coronary. The latter value approaches, equals or exceeds the aortic systolic pressure simultaneously recorded. This pressure increment has its source largely in the other non-occluded coronaries.

The intravenous pressure (in a coronary vein or in the coronary sinus) rises from control values of say 10/2 mm Hg. to figures which during systole approach or exceed the aortic systolic and whose diastolic value reaches 20 to 40 mm Hg. This pulse is patterned in time and contour after the peripheral coronary pressure while its ordinate values are generally slightly greater.

The retrograde blood flow is markedly elevated in the ramus descendens anterior from a maximum normal value approximating 1 cc per minute to values as high as 39 cc per minute. The maximum peripheral flow is reached in from 10 to 30 minutes and following sinus release does not immediately return to control backflow figures. This increase does not occur in the right coronary. The blood is highly unsaturated, containing only 3-4 volumes percent oxygen, while the coronary sinus approximates 8 volumes percent oxygen.

Such a volume of blood with its low oxygen content is not sufficient for a region of myocardium whose coronary has been occluded, for myographic records show systolic extension within 2-3 minutes after occlusion of a major left coronary ramus, while the peripheral coronary is either closed or allowed to bleed.

In view of these findings it is believed that after sinus blockage an additional mean volume of blood coming largely from the coronary arteries is trapped in the venous system, thus raising the pressure in both venous and arterial vascular beds. However, there are potent drainage channels still remaining and presumably largely venous, since the left coronary inflow is only moderately reduced and the

[4] Gregg, D. E., Green, H. D., and Wiggers, C. J., *Am. J. Physiol.*, 1935, **112**, 362.

myocardium fails to contract following left coronary artery ligation. Consequently, as a result of the combined occlusion of the sinus and a left coronary ramus, dynamic conditions are created, such that there should be a sizable blood flow from non-occluded coronaries through their capillary beds into the large veins and then by Thebesian and arterioluminal vessels into the heart cavities.

If the peripheral coronary artery is closed (not allowed to bleed) it is believed that no adequate anatomical circuit exists to route this venous blood through the capillary bed of the occluded coronary, whereas if the peripheral coronary is opened a portion of this blood is routed in retrograde fashion from the venous system through the capillary bed of the occluded artery. Since ligation of the cardiac veins does reduce considerably the normal left coronary inflow and does not prevent failure of contraction in a myocardial area whose normal blood supply has been acutely removed, such a procedure cannot be regarded as a method of choice for encouraging the blood supply to a potentially infarcted area.

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Inhibitory Action of Narcotics on the Histamine Contraction of Plain Musculature.

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As was first shown by Besredka[1] narcosis may prevent fatal anaphylactic shock in guinea pigs. This effect of the narcotic is not due to its action on the central nervous system as presupposed by Besredka, but is probably attributable to its peripheral action on the bronchial musculature (Farmer[2]). In *in vitro* experiments I[3] was able to demonstrate that narcotics (urethane, chloralose) inhibit anaphylactic contraction of sensitized uterine strips of guinea pigs without inhibiting desensitization of the muscle.

Dale's[4] theory of anaphylaxis assumes that anaphylactic contraction of plain musculature is caused by liberated histamine or "histamine-like" substances. In my experiments (l. c.) anaphylactic con-

1 Besredka, *Ann. de l'Inst. Pasteur*, 1907, **21**, 957.

2 Farmer, *J. Immunol.*, 1937, **32**, 195.

3 Farmer, *J. Immunol.*, 1937, **33**, 9.

4 Dale, *Lancet*, 1929, I, 1179, 1233, 1285.