to be evidence of morbidity. The British are slightly more critical, their standard being 100 F. on two successive days other than the first. De Lee has expressed the opinion that a temperature of 100 F. on any day should be considered as a sign of morbidity. Perhaps he is somewhat too severe in asking for a classification of all first day temperatures as evidence of infection, and therefore of morbidity. It is generally believed that the usual response to a sudden flooding of the

TABLE 2.—*Maternal Morbidity in Four Hundred Patients*

American Standard*		British Medical Association Standard, Additional		De Lee Standard: First Day Only, Additional		De Lee Standard: One Day Only (Other Than First) Additional		Total for All Standards	
Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
15	3.75	11	2.75	16	4	38	9.5	80	20

* The corrected morbidity rate according to American standards was 2.25 per cent (nine patients).

system with a foreign protein is that of an initial rise in temperature, and such a condition may prevail at the time of parturition.

Table 2 shows the uncorrected morbidity rate in the present series according to the accepted American, British and De Lee standards. De Lee's standard has been subdivided to show those patients with only first day temperatures of 100 F. or over, and those with one day of temperature of 100 F. or over occurring on any other day but the first.

The morbidity in this series, judged by any or all of the aforementioned classifications, I feel, compares quite favorably with that in other series. Among the fifteen febrile patients, as judged by the American standard, were two with acute mastitis, one with acute pharyngitis and rhinitis, one with influenzal pneumonia (admitted in that state), one with pyonephrosis, one with toxemia of pregnancy, admitted with a temperature of 101.4 F., five with sapremia, three with acute endometritis and one with an infected perineal wound. The first five of these patients obviously did not show an obstetric morbidity, and the sixth, although having an obstetric complication, was admitted in a febrile state. If these patients are omitted from the series, the corrected morbidity is only 2.25 per cent. I believe that this is a fairly good showing.

COMMENT

The immediate administration of a suitable oxytocic that will maintain the tonicity of the uterine musculature may obviate the low grade, morbid temperatures which so often are classified as sapremia. In most delivery rooms it is customary to administer from 0.5 to 1 cc. of solution of pituitary after expulsion of the placenta. It is known that the action of solution of pituitary is short lived. If a reasonable tonus of the uterus can be maintained for an extended period of time, the chances for the accumulation of blood clot and decidual remnants is greatly diminished.

That a mildly tonic contraction of the uterus is maintained for a longer period with 1 cc. of ergotamine tartrate than with solution of pituitary is evidenced by a comparison with the series of 167 patients who received 1 cc. of solution of pituitary hypodermically immediately after expulsion of the placenta. None received the ergot derivative in this stage. The average height of the fundus in all of these cases on the first day was 15.1 cm. On the other hand, in the current series, wherein 1 cc. of ergotamine tartrate was almost universally replaced by solution of pituitary, the average height on the first day post partum was 11.8 cm.

Theoretically, at least, it would seem that the cultural mediums in the uterine canal and the gaping sinuses are reduced to a minimum. The continuation of the mildly tonic contraction is maintained during the succeeding three days by the oral administration of the oxytocic in small doses.

The absence of foul lochia bespeaks the absence of sapremia. It would seem that with the decrease in the size of the uterus, undoubtedly at the expense of the cavity, both the "soil and the pathway" for the propagation of an infection are materially reduced, thus lessening at least one possible source of infection. In the present series it was possible to reduce the morbidity in this way to 2.25 per cent.

CONCLUSION

Four hundred parturient patients were given ergotamine tartrate hypodermically immediately after delivery of the placenta and orally for the ensuing three days. It is hoped that a means has been presented for reducing the controllable maternal morbidity. It is felt that any method that will help, however slightly, to carry a parturient woman safely through the lying-in period is worthy of a trial.

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THE SPECIFIC TREATMENT OF LOBAR PNEUMONIA

A STATISTICAL REVIEW

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The specific treatment of lobar pneumonia was attempted as early as 1897 by Washbourn.[1] His and other early attempts were not successful, because the serums were not made and given on a type specific basis. Since 1913 Cole [2] has used an unconcentrated horse serum in type I pneumonia with a mortality of 10 per cent. This unconcentrated serum, however, has not become popular because of its tendency to produce reactions.

In 1921 Huntoon [3] produced a serum-free solution of pneumococcus antibodies in modified Ringer's solution. The potency of this product is such that it protects mice against 1,000,000 lethal doses of a virulent culture of pneumococcus type I. It is somewhat less protective against type II and protects against only 100,000 lethal doses of type III. It contains only 0.035 mg. of nitrogen per cubic centimeter. Recently the manufacturers have discontinued the use of pneumococcus type III in the preparation of this solution.

In 1924 Felton [4] described a method of concentrating and refining antipneumococcic horse serum and has subsequently improved the method. This product represents approximately a tenfold concentration of

1. Washbourn, J. W.: Antipneumococcus Serum, Brit. M. J. **1**: 510 (Feb. 27) 1897.
2. Cole, Rufus: Serum Treatment in Type I Lobar Pneumonia, J. A. M. A. **93**: 741 (Sept. 7) 1929.
3. Huntoon, F. M.: Antibody Studies, J. Immunol. **6**: 117 (March) 1921.
4. Felton, L. D.: A Study of the Isolation and Concentration of the Specific Antibodies of Antipneumococcic Sera, Boston M. & S. J. **190**: 819 (May 15) 1924.

the serum with a corresponding increase in its potency. It consists largely of a portion of the globulins. Its nitrogen content is about the same as that of whole serum. This concentrate is marketed in units, a Felton unit being that amount which will protect a mouse against 1,000,000 lethal doses of a virulent culture of pneumococci. As different lots of serum vary considerably in antibody content, it is important to estimate doses in units and not in cubic centimeters. Some

TABLE 1.—*The Effect of Pneumococcus Antibody Solution in Lobar Pneumonia in Adults* *

Types	Treated	Mortality, per Cent	Controls	Mortality, per Cent	Mortality Reduced, per Cent	Lives Saved per 100 Cases
I	186	14	169	23	39	9
II	98	29	71	38	24	9
III	91	36	65	42	12	5
IV	179	12	127	24	50	12
	554	20	432	29	28	9

* Combined reports of Cecil and Larsen, Conner, Belk and Sharpe.

reports, however, express doses in cubic centimeters. The reason for this is, of course, that the liability to the production of reactions is in proportion to the amount of serum given at one time and not to the unit value.

The results obtained with specific therapy are shown in the tables. These include all the reports in American and foreign literature up to January 1935 that lend themselves to statistical study.[5] The several series differ somewhat in doses administered, in the method of selecting cases for treatment, and in the method of selecting control groups. They are, however, fairly comparable and demonstrate the value of the method under average conditions. "Serum" as used in the text and the tables indicates concentrated serum. In most instances serum was prepared by Felton's method.

In table 1, pneumococcus antibody solution is seen to be definitely beneficial in type I pneumonia, less so in type II, and of little or no value in type III pneumonia. Surprisingly, it is more effective in group IV than in type I infections. This is due, probably, to a content of group specific antibodies in addition to its type specific antibodies. The observation is a most important one, particularly since it is demonstrated in all of the three series. This of course has practical importance.

In table 2, antipneumococcic serum is seen to reduce the mortality in type I lobar pneumonia by 40 per cent and to save ten lives per hundred cases. It is of less value in type II but of distinct benefit in types VII and VIII. There is no apparent reason for the rather wide difference in the results of different observers. This series covers a wide range of conditions, some unfavorable, such as the beginning of treatment late in the disease, and the inclusion of a number of old patients. The report of Heffron and Anderson is of special interest, as it represents the work of sixty-three practitioners in the state of Massachusetts, under the direction of the department of health, and shows that the specific treatment of lobar pneumonia is not a method requiring specially organized hospital services.

When fatalities in the treatment group are considered, it is found that many of these patients were suffering from morbid processes other than the pneumonia. Senility, alcoholism, heart disease and other

TABLE 2.—*The Effect of Concentrated Antipneumococcus Serum in Lobar Pneumonia in Adults*

	Dates	Treated	Mortality, per Cent	Controls	Mortality, per Cent	Mortality Reduced, per Cent	Lives Saved per 100
		Type I					
Cecil and Plummer	24-29	239	20	234	31	35	11
Park, Bullowa and Rosenbluth	26-28	109	17	105	31	45	14
Baldwin	26-30	19	5	20	25	79	20
Bullowa	28-32	309	16	54	29	45	13
Sutliff and Finland	29-32	75	16	76	37	56	21
Medical Research Council	30-33	184	10	301	15	34	5
Heffron and Anderson	31-33	188	11	85	26	59	15
Leys	32-33	15	26	15	20	+33	−7
		1,138	15	890	25	40	10
		Type II					
Cecil and Plummer	24-30	252	40	253	46	12	5
Park, Bullowa and Rosenbluth	26-28	56	23	61	30	23	7
Baldwin	26-30	35	26	29	52	50	26
Bullowa	28-32	76	30	84	43	30	13
Finland and Sutliff	29-32	46	20	81	40	50	20
Medical Research Council	31-33	164	20	305	27	25	7
Heffron and Anderson	31-33	48	25	...	..	..	..
		677	30	799	37	19	7
		Type VII					
Bullowa	28-31	15	7	85	26	74	19
		Type VIII					
Bullowa	33	37	5	85	16	67	11

complicating conditions were of frequent incidence, and many of the cases were treated late in the disease. Possibly a fairer picture of specific treatment is obtained by the study of its results under more favorable conditions, as shown in table 3.

Table 3 shows an average reduction in mortality of about 50 per cent, with a saving of twenty lives per hundred cases. The 78 per cent reduction in mortality in type II pneumonias, with a saving of fifty lives per hundred cases in the selected series of Cecil and Plummer, is worthy of special mention. This table illustrates the importance of beginning specific treatment as early after the onset of pneumonia as possible.

5. These include:

Cecil, R. L., and Larsen, N. P.: Clinical and Bacteriological Study of One Thousand Cases of Lobar Pneumonia, J. A. M. A. **79:** 343 (July 29) 1922.

Conner, L. A.: Experiences in the New York Hospital with the Treatment of Lobar Pneumonia by a Serum-Free Solution of Pneumococcus Antibodies, Am. J. M. Sc. **164:** 832 (Dec.) 1922.

Belk, W. P., and Sharpe, J. S.: Pneumococcus Antibody Solution in the Treatment of Lobar Pneumonia, Am. J. M. Sc. **187:** 844 (June) 1934.

Cecil, R. L., and Plummer, Norman: Pneumococcus Type I Pneumonia, J. A. M. A. **95:** 1547 (Nov. 22) 1930.

Park, W. H.; Bullowa, J. G. M., and Rosenbluth, M. B.: The Treatment of Lobar Pneumonia with Refined, Specific, Antibacterial Serum, J. A. M. A. **91:** 1503 (Nov. 17) 1928.

Baldwin, H. S.: The Specific Therapy of Pneumococcus Type I and Type II Pneumonia, Am. J. M. Sc. **181:** 788 (June) 1931.

Bullowa, J. G. M.: Studies in the Serum Treatment of Pneumonia, New York State J. Med. **33:** 13 (Jan. 1) 1933.

Sutliff, W. D., and Finland, Maxwell: Type I Pneumococcus Infections with Special Reference to Specific Serum Treatment, New England J. Med. **210:** 237 (Feb. 1) 1934.

Report of the Therapeutic Trials Committee of the Medical Research Council: The Serum Treatment of Lobar Pneumonia, Lancet **1:** 290 (Feb. 10) 1934.

Heffron, Roderick, and Anderson, G. W.: Two Years' Study of Lobar Pneumonia in Massachusetts, J. A. M. A. **101:** 1286 (Oct. 21) 1933.

Leys, Duncan: Observations on the Serum Treatment of Type I Lobar Pneumonia, Lancet **2:** 748 (Sept. 30) 1933.

Cecil, R. L., and Plummer, Norman: Pneumococcus Type II Pneumonia, J. A. M. A. **98:** 779 (March 5) 1932.

Finland, Maxwell, and Sutliff, W. D.: The Specific Serum Treatment of Pneumococcus Type II Pneumonia, J. A. M. A. **100:** 560 (Feb. 25) 1933.

Bullowa, J. G. M.: The Therapeutic Value of Specific Type VII (Cooper) Antipneumococcic Serum, Libman Ann. Vol. **1:** 283 (Jan. 1) 1933.

Bullowa, J. G. M.: Therapeutic Pneumococcus Type VIII (Cooper) Serum, J. A. M. A. **102:** 1560 (March 12) 1934.

It also shows the value of this therapy in septicemia, owing to its demonstrated power of sterilizing the blood stream.

In addition to mortality statistics, all observers mention definite clinical benefits in many cases, such as

TABLE 3.—*Specific Treatment of Lobar Pneumonia in Adults: Selected Series*

	Treated	Mortality, per Cent	Controls	Mortality, per Cent	Mortality, Reduced, per Cent	Lives Saved per 100
Type I. Bacteremias—Serum						
Bullowa	96	39	10	80	51	41
Sutliff and Finland	38	26	42	71	63	45
Park, Bullowa and Rosenbluth	28	36	28	71	49	35
Medical Research Council	28	17	23	17	0	0
	190	33	103	60	45	27
Type II. Bacteremias—Serum						
Bullowa	26	69	35	77	10	8
Finland and Sutliff	11	55	29	69	20	14
Park, Bullowa and Rosenbluth	14	50	11	82	37	32
Medical Research Council	18	39	22	50	22	11
	69	55	97	69	20	14
Type I. 72 Hour Series—Serum						
Cecil and Plummer	103	12	97	27	56	15
Type I. 96 Hour Series—Serum						
Sutliff and Finland	75	16	76	37	56	15
Type II. 72 Hour Series—Serum						
Cecil and Plummer	21	14	20	65	78	51
Type I. 48 Hour Series—Pneumococcus Antibody Solution						
Cecil and Plummer	56	9	68	24	62	15

reduction in temperature and in pulse and respiration rates, correction of cyanosis and delirium, early crises and shortening of the duration of illness. The blood

TABLE 4.—*Specific Treatment of Lobar Pneumonia by Intramuscular and Subcutaneous Administration*

		Mortality Treated, per Cent	Mortality Controls, per Cent	
Cecil and Baldwin	Pneumococcus antibody solution subcutaneously	34	35	All types; late and early cases
Cecil and Baldwin	Pneumococcus antibody solution subcutaneously	24	39	All types; 48 hour series
Oliver and Staller	Pneumococcus antibody solution subcutaneously	17	24	All types; 100 hour series
Becker	Serum intramuscularly	8	20	Type I; quinine in all cases
		15	30	Type II; quinine in all cases
Belk and Sharpe	Pneumococcus antibody solution intramuscularly			Clinical improvement in children
Nemir	Serum intramuscularly			Clinical improvement in children

stream is sometimes sterilized, and extensions of the infection are often prevented. The incidence of complications, however, appears to be about the same in the treated and the untreated patients.

These statistics are based on intravenous serum therapy. The possibility of administering specific antipneumococcus therapy by intramuscular and subcutaneous injections is an attractive one because of the greater ease as compared to the intravenous route. Curphy and Baruch have shown that intramuscular injections are as effective as the intravenous in rabbits suffering with pneumococcic skin infections.[6] On the other hand, Rhoades[7] found that pneumococcus antibodies injected subcutaneously often failed to appear in the circulation. In table 4 are given the results of treatment by these routes.[8]

Table 4 shows that some benefit doubtless results from intramuscular and subcutaneous injections of specific preparations, but this is clearly smaller than after intravenous administration. At present these substitute methods would seem to be justified only when it is impossible to use the intravenous route.

TABLE 5.—*Dosage in Specific Antipneumococcus Therapy*

Day of Disease on Which Treatment Is Begun	Concentrated Serum: Cubic Centimeters of Serum First 24 Hours	Units
1–24 hours	5, 25	90,000
24–48 hours	5, 25, 45	225,000
48–72 hours	5, 25, 45, 45	360,000
72 hours	5, 25, 45, 45, 45	495,000

Given at intervals of two hours
Amounts on subsequent days as indicated clinically
Pneumococcus antibody solution, from 50 to 100 cc. three times a day

Table 5 gives a conventional scheme of dosage, that for serum being suggested by Sutliff and Finland.[9] The first consideration of dosage is to administer serum, or antibody solution, as early in the disease and in as large amounts as is safely possible. Beneficial effects are not experienced until the soluble specific substance in the patient's blood is neutralized by an excess of antibodies. Patients first treated late in the infection, those with septicemia and those with type II pneumonia, require larger doses to effect this result. After the first day's dosage, serum is given either in the same amounts (if no improvement has taken place) or in smaller amounts, until the temperature falls and remains near normal. Armstrong and Johnson[10] feel that if no benefit is observed after seven doses of 20 cc. each (given at intervals of from six to eight hours) it is probably not worth while to continue the administration.

TABLE 6.—*Reactions Following Specific Antipneumococcus Therapy*

Reaction	Concentrated Serum	Antibody Solution
Severe allergic	Very rare	None
Mild allergic	5-15%	None
Thermal	10-20%	Frequent to rare
Serum sickness:	25-30%	None

Table 6 gives a summary of the incidence of reactions as recorded in the several reports. The figures are only approximate. When precautions are taken to

6. Curphy, T. J., and Baruch, H. B.: The Therapeutic Value of Intramuscular Dosage of Type I Pneumococcus Antisera, J. Exper. Med. **55**: 925 (June) 1932.

7. Rhoades, D. R.: The Fate of Pneumococcic Protective Bodies When Injected into Normal Animals and Man, Bull. 141, Hyg. Lab., U. S. P. H. S., April 1925.

8. Cecil, R. L., and Baldwin, H. S.: The Treatment of Lobar Pneumonia with Subcutaneous Injections of Pneumococcus Antibody Solution, J. Pharmacol. & Exper. Therap. **24**: 1 (Aug.) 1924. Oliver, W. W., and Staller, E. A.: Notes on the Therapeutic Value of Pneumococcus Antibody Solution Subcutaneously Administered in Lobar Pneumonia, Arch. Int. Med. **35**: 266 (Feb.) 1925. Becker, Hellmuth: Beitrag zur Serumbehandlung der Pneumonie, München. med. Wchnschr. **81**: 1487 (Sept. 28) 1934. Nemir, R. L.: The Treatment of Pneumonia in Infants and Children with Antipneumococcus Serum, J. Pediat. **3**: 827 (Dec.) 1933.

9. Sutliff, W. D., and Finland, Maxwell: Type I Pneumonia Treated with Concentrated Pneumococcic Antibody (Felton), J. A. M. A. **96**: 1465 (May 2) 1931.

10. Armstrong, R. R., and Johnson, R. S.: Homologous Antipneumococcal Serums in the Treatment of Lobar Pneumonia, Brit. M. J. **1**: 931 (May 30) 1931.

make skin and ophthalmic tests, and to refuse serum to those with positive ophthalmic tests and also to those with a history of allergy to horses, the question of reactions appears not to be a serious one. These tests are made with a 1 : 10 dilution of concentrated serum or better, possibly, with a 1 : 10 dilution of whole normal horse serum, as the concentrated serum is said to give false positive skin tests. The mild allergic reactions are easily controlled with epinephrine. Only one allergic death is reported. The antibody solution does not cause any allergic reactions and has been given successfully to known allergic individuals.

Reports of specific antipneumococcus therapy in children are few. Nemir observed its effects in both lobar pneumonia and bronchopneumonia produced by pneumococcus types I to XXII inclusive. The mortality in the entire group was reduced from 16.6 per cent to 9.8 per cent.

The specific treatment of pneumococcic infections is probably destined to increase in popularity. Serums will doubtless be still further refined, with the result that reactions will be largely eliminated and larger initial doses will be possible. Potent serums for the recently identified types of pneumococci will in all probability soon be available commercially. The demonstrated merit of specific therapy in lobar pneumonia would seem to justify its use in bronchopneumonia and other pneumococcic infections, such as mastoiditis, thus giving it a wider field of usefulness.

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ANGINAL SYMPTOMS ASSOCIATED WITH CERTAIN CONSTITUTIONAL DISEASES

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Although angina pectoris has been recognized for nearly two centuries, its pathologic physiology continues to be obscure. Whatever theory is subscribed to, whether the coronary, the aortic or the myocardial, it is generally agreed that the actual attack is precipitated by a demand on the heart which for one reason or another cannot be met.

Keefer and Resnik [1] have proposed on excellent theoretical and factual grounds that anoxemia of the myocardium, however brought about, is the fundamental pathologic condition.

In the past few years I have had occasion to see a number of patients whose presenting complaint was typical angina or attacks indistinguishable from it. Further studies of these cases have revealed various constitutional diseases, the adjustment of which has either improved or done away with the anginal attacks. This relief allows of some interesting speculations on the cause of angina.

In the following case reports, data that have not seemed pertinent have been purposely omitted for the sake of brevity.

REPORT OF CASES

CASE 1.—M., a man, aged 53, a clerk, admitted to St. Luke's Hospital, March 17, 1932, complained of nervousness and discomfort about the heart. About two and one-half years before admission he became nervous and noticed that his heart was beating very rapidly, and at the same time his wife thought that his eyes appeared more prominent than usual. In spite of an excellent appetite he lost weight. About a year before admission he began to suffer with precordial pain accompanied by shortness of breath, which was brought on by exertion and relieved by rest. There was no edema.

During the next year all the symptoms, including the frequency of precordial discomfort, became more marked. The morning before admission, while dressing, he was seized by a sudden severe pain in the precordial region, which radiated down his left arm. There was extreme shortness of breath and the patient felt that he was in a very precarious condition. A hypodermic of morphine was necessary for relief.

On admission the temperature was 99.6 F., the pulse 140, and the blood pressure 140 systolic, 60 diastolic. The skin was moist and clammy and there was definite exophthalmos. The thyroid was moderately and symmetrically enlarged and there was a fine tremor of the extended fingers. The heart showed an apparent moderate enlargement. The rhythm was very rapid and irregular, strongly suggesting auricular fibrillation. A systolic murmur was heard over the entire precordium, loudest at the apex. The lungs were clear, the liver was not palpable and there was no edema of the feet or ankles. An electrocardiogram confirmed the impression of auricular fibrillation.

A diagnosis of exophthalmic goiter with cardiac enlargement and auricular fibrillation was made. Of course a strong suspicion of angina pectoris was entertained.

During the next several weeks the basal metabolic rate varied from plus 35 to plus 40, and there was considerable general improvement on the preliminary medical management. The cardiac rhythm became regular, with an average pulse rate of 88. April 18, one month after admission, a partial thyroidectomy was done. Recovery was uneventful. He was last seen in July 1933, somewhat more than a year after operation. He had returned to work and was in an excellent general physical condition. There had been no further anginal attacks.

CASE 2.—A., a man, aged 63, a clerk of the court, seen in the McGuire Clinic, Jan. 14, 1929, complained of numbness of the hands and toes, difficulty in walking, and attacks of precordial pain and oppression brought on by exertion. The present illness dated from about three years before, when he began to have attacks characterized by pain and discomfort in the left side of the chest, at times radiating down the left arm, and associated with a feeling of tightness in the chest and shortness of breath. These attacks were brought on by some unusual exertion and relieved by rest. Recently they had become more frequent, and much less exertion was required to precipitate them. During the past six weeks he had noticed numbness and tingling of the hands and toes and difficulty in walking, which had grown progressively worse. His hands had become so involved that he could not sign his name legibly.

On examination the temperature was 98 F., the pulse 78, and the blood pressure 150 systolic, 82 diastolic. The gait showed marked spastic ataxia. The skin was somewhat sallow. The hands were slightly edematous and distinctly ataxic. The apex of the heart was normally located and there was a systolic murmur heard at the base. There was a slight sinus arrhythmia. The lungs were clear; the liver was not palpable. Generally increased muscular tonus, anesthesia of the extremities, and loss of vibratory sense were present.

Examination of the blood revealed hemoglobin 75 per cent and red blood cells, 2,820,000, with a color index of 1.3. There was marked macrocytosis with hyperchromia. Poikilocytosis, anisocytosis and polychromatophilia were present. The reticulated count was 1.1 per cent; leukocytes, 8,000; polymorphonuclear leukocytes, 66 per cent; lypmhocytes, 27 per cent, and eosinophils, 5 per cent. The gastric analysis showed an absence of free hydrochloric acid, with a total acidity of 6. The blood Wassermann reaction was negative. The electrocardiogram showed a sinus bradycardia with a rate of 50 but was otherwise normal. The basal metabolic rate was minus 15. Two days later the blood work was repeated with approximately the same results.

A diagnosis of pernicious anemia and coronary disease was made.

From the McGuire Clinic and St. Luke's Hospital.
Read before the Richmond Academy of Medicine, Feb. 12, 1935.

1. Keefer, C. S., and Resnik, W. H.: Angina Pectoris; Syndrome Caused by Anoxemia of Myocardium, Arch. Int. Med. **41**: 769 (June) 1928.