Groedel[67] found that the Nauheim baths did not cause an injurious increase of the blood-pressure when their temperature was almost indifferent, *i.e.*, 92° to 93° F. (33.3° to 33.9° C.), the primary contraction of the cutaneous vessels passing off quickly.

An essential feature of the treatment is to avoid the retention in the intestinal canal of dejecta capable of causing autointoxication. *Saline cathartics,* the best of which is the citrate of magnesia, should be taken periodically if needed. In some cases the accumulation of toxics is mainly due to hepatic torpor; a purgative dose of *blue mass,* followed by a saline purgative, given at intervals of two weeks, aids materially the curative process, by freeing the intestinal canal of any accumulation of excreta, thus obliterating a common source of autointoxication.

Measures which Counteract Directly the Morbid Process.—The beneficial action of *thyroid gland* is accounted for by the controlling action which large doses of this agent have over the adrenal system.* By increasing the sensibility of the depressor nerve (Cyon) owing to the excess of thyroidase it contributes to the blood, thyroid gland causes constriction of the arterioles through which the anterior pituitary and the thyroid apparatus are supplied with blood.* The supply of adrenoxidase (besides thyroidase) being diminished, the metabolic activity in the vascular walls is reduced,* and the chief pathogenic process is thus controlled.*

The large doses of thyroid gland that have been used are unnecessary.* When the vascular tension is high, the blood already contains a large proportion of thyroidase and small doses sometimes suffice to raise the proportion of the latter to the point at which it will control the depressor, and through it reduce the functional activity of the adrenal system.* The arterial tension should be the guide, and starting with 5 grains (0.3 gm.) three times daily (taken during meals), the dose should be increased or decreased according to the condition of the pulse and the resistance of the arterial system in general. Large doses may cause a sufficiently violent fall of the blood-pressure by causing excessive constriction of the pituitaral and thyroidal arterioles* through the depressor, that cardiac arrest may occur.

Lancereaux[68] observed marked benefit in a well-defined case in which he gave 30 to 45 grains (2 to 3 gms.) daily. The arterial tension was lowered and the hard, calcareous arteries seemed to undergo resolution. Geo. Oliver, according to Barr,[69] showed several years ago that thyroid extract caused dilation of the arteries, and Barr, in experiments upon himself, found that it increased tissue metabolism. M. Allen Starr, as previously stated, has repeatedly observed the "constant therapeutic effect of the administration of thyroid extract in lowering tension."

* *Author's conclusion.*
[67] Groedel: *Loc. cit.*
[68] Lancereaux: Semaine méd., Jan. 4, 1899.

Huchard[70] in a paper on the use of hyptotensive medication writes, alluding to thyroid extract: "The latter has been classed by Livon among the glands which have a vasoconstrictor action, but its vasodilator and hypotensive action has been demonstrated by the experiments of Oliver and Schäfer, Haskovec, Cunningham, Cyon, Gley and Langlois, Guinard and Martin." I have shown that these two divergent views are both sound, since *small* doses *raise* the vascular tension by exciting the pituitary body, while *large* doses *lower* the blood-pressure by depressing the pituitary. Alluding to the latter effect, Huchard also says: "One can thus understand how and why thyroid extracts, and especially iodothyrin, were able to benefit, according to Lancereaux and Paulesco, patients suffering from scleroderma, vasomotor disorders of the extremities and arteriosclerosis. But their hypotensive action is brusque and sudden; it may even be attended with asthenia and cardiac collapse, which is a great inconvenience, the purpose of hypotensive medication being always to relieve the heart without weakening it."

Acting in the same manner, but less energetically, are the *iodides,* which have been extensively used. Beginning with 5 grains (0.3 gm.) three times daily, in a tumberful of water, after meals, the dose may gradually be increased until 15 grains (1 gm.) are given. The remedy should be taken during a prolonged period, suspending its use one week every month. It is efficacious irrespective of any syphilitic taint.*

E. Romberg[71] holds that the efficacy of small doses of potassium iodide has been established. James Barr[72] says that iodine is often more valuable than thyroid. Combemale[73] considers potassium iodide the remedy "*par excellence*" in arteriosclerosis. Milk seems to facilitate the tolerance of this drug, and is at the same time the best vehicle for its administration. Jodlbauer[74] ascribes its beneficial action to the fact that it distinctly dilates the arterioles. That the action of potassium iodide corresponds with that of thyroid extract is well shown by the following quotation from a paper by Cummins and Stout[75] on experimental arteriosclerosis: "It has already been shown by Prévost, Binet and others, that the iodide in large doses produced diminished vascular tension, and is efficacious in overcoming the spasmodic condition of the sclerosed vessels."

In some cases the persistence of a high degree of arterial tension demands prompt action. An indication of this is insom-

* *Author's conclusion.*
[69] Barr: *Loc. cit.*
[70] Huchard: Revue de thérap. méd-chir., vol. lxx, p. 433, 1903.
[71] E. Romberg: Deut. med. Woch., Bd. xxxii, S. 1377, 1905.
[72] James Barr: *Loc. cit.*
[73] Combemale: L'echo méd. du Nord., vol. v, p. 69, 1901.
[74] Jodlbauer: Münch. med. Woch., Bd. xlix, S. 653, 1902.
[75] Cummins and Stout: Univ. of Penna. Med. Bull., July, 1906.

nia, due to cerebral hyperæmia—a condition which, in itself, entails the danger of cerebral hæmorrhage. The sensibility of the vascular centers must then be reduced artificially.. *Nitroglycerin* is the best remedy for this purpose, $^1/_{120}$ grain to $^1/_{60}$ grain (0.00054 to 0.001 gm.) being taken at bedtime and kept up a week, unless headache or nausea supervene. When it loses its effects, the other nitrites may be tried. The *bromides* or *chloral hydrate,* or better combined, giving 10 grains (0.6 gm.) on retiring, may be used as substitutes. The *bromides* alone in 20-grain (1.3 gm.) doses, as needed, are very efficacious when a high blood-pressure and headache point to the presence of considerable cerebral hyperæmia.*

J. M. Anders[76] states that sphygmographic tracings indicate the vasodilator effects of nitroglycerin in arteriosclerosis and that the drug is indicated and most effective in early cases when there is a marked increase in the arterial pressure and the heart is hypertrophied. H. J. Campbell[77] holds that when the heart is hypertrophied and its muscle beginning to fail, the main indication is undoubtedly to save the work of the heart as much as possible. The regular administration of small doses of nitroglycerin is beneficial; but massage and restricted movements are strongly contraindicated, as is also the use of digitalis, strychnine or other heart tonics. Attention to the bowels, as in all cases of cardioarterial disease, is of the first importance. The value of the bromides in such conditions I have been able to appreciate in my own cases.

The *second stage* includes the period during which the functions of various organs, the heart, the kidneys, etc., are compromised by marked organic lesions. Thus atheroma of the base of the aorta often extends to the aortic valves, causing them to fuse together or to adhere to the aortic walls, thus causing insufficiency or stenosis of the aortic orifice. Again, when the small branches of the coronaries are alone sclerosed, degeneration of the areas supplied by the distributing capillaries follows and the muscular fibers destroyed are replaced by fibrous tissue. The remaining muscular elements taking on more work to compensate for the loss, they are overfed and become hypertrophied, the whole organ becoming through this process more or less enlarged. The kidneys, we have seen, are the seat of a marked irritation which ultimately leads to the condition of granular kidney. The liver, the brain, the pancreas and all other organs,

* *Author's conclusion.*
[76] J. M. Anders: N. Y. Med. Jour., June 25, 1904.
[77] H. J. Campbell: Brit. Med. Jour., Oct. 12, 1901.

in fact, may bear the brunt of the disease and give rise to special symptoms which are grafted upon the morbid phenomena of the general disease, and require measures directed to these organs, as illustrated below in the case of the heart.

The remedial measures recommended for the first stage are all applicable in the second. When seen late, however, the hypertrophied heart may show evidences of degeneration, especially of the right ventricle. Here the judicious use of *digitalis* becomes necessary, provided the arterial tension be normal or subnormal, which is often the case when the disease is advanced. Digitalin, $^1/_{10}$ grain (0.0065 gm.) twice daily, is effective under these conditions. If the tension rises, *nitroglycerin, sodium nitrite or erythrol tetranitrate* may be used to counteract this effect. We thus support the heart and simultaneously avoid unusual resistance of the blood-column to the contractions of its walls.

The combined use of digitalis and the nitrites was introduced by Huchard and has been advocated by Balfour, DaCosta and others, but it must be resorted to with circumspection. Aged patients seldom bear digitalis well. Delancey Rochester[78] contends that it is always a dangerous drug in arteriosclerosis. Combemale[79] states that in the last stages of arteriosclerosis, digitalis and digitalin or sparteine are the only resources we have left to produce even a palliative effect.

ANGINA PECTORIS.

SYNONYMS.—*Stenocardia; Breast-pang.*

Definition.—A paroxysmal disorder of the heart the characteristic symptom of which, severe pain in and around this organ, is due to an excessive and violent influx of arterial blood into the myocardium and its nervous elements. This flooding of the cardiac muscle is due, in turn, to inability of the coronary arteries, when atheromatous, to contract sufficiently under the influence of their vasomotor nerves to prevent it when the blood-pressure of the body at large becomes high, owing to the presence, in the blood, of waste-products in sufficient quantities to excite the vasomotor center.*

Symptoms.—The most prominent symptom, pain, may be exeruciating and comes on suddenly, often after mental excitement, a copious meal, exposure, a muscular effort, etc. It be-

* *Author's definition.*
[78] Delancey Rochester: Med. News, Nov. 2, 1901.
[79] Combemale: *Loc. cit.*

gins behind the sternum or in the heart, this organ feeling as if violently constricted, compressed or stabbed, and extends into the neck, the back, the shoulders, down the inner aspect of the left arm, often to the wrists and finger-tips. In some cases the pain is even more diffuse, radiating to the head and both shoulders, the trunk and lower extremities, and may be followed by anæsthesia of the left hand or arm, preceded by tingling. The face may be pale—ashen-gray rather—or flushed, and is usually covered with sweat. The features betray the intense agony to which the patient is subjected and fear of impending death. The cardiac action is often regular, but in some cases the gallop rhythm occurs or the action is turbulent and irregular; then weak and distant. The pulse varies considerably, being at one time slow and tense, then fast, and finally irregular and even imperceptible, to recover again and resume its normal action. Often, however, the pulse shows but little, if any, change. Dyspnœa, strictly speaking, is seldom present, but the respiration is shallow, a deep breath being taken at intervals. In the exceptions, however, typical asthma may be present, including the piping râles during expiration. Consciousness is seldom lost except in the final attack. In favorable cases the pain may cease as abruptly as it began, gaseous eructations and a copious flow of urine marking the end of the attack.

In most cases, the disease begins insidiously, the attacks being at first slight and occurring at long intervals. Gradually, however, they become more severe and appear more frequently until death occurs—not always in the midst of a paroxysm, but suddenly and without warning. In other cases the attacks follow one another in rapid succession; in a third group death occurs during the first paroxysm.

Pathogenesis.—The primary cause of angina pectoris is an organic lesion of the coronary arteries. It is not, however, as now believed, due to obstruction or stenosis of these vessels, but, on the contrary, to their inability to respond to the constrictor impulses of the vasomotor nerves distributed to them, and through which the volume of blood supplied to the heart-wall is regulated.*

* *Author's conclusion.*

As I have pointed out elsewhere,[80] the physiological rôle of the vasomotor and sympathetic network supplied to the coronaries and their branches is that fulfilled by these nerves elsewhere, viz., to constrict their muscular layer and thus reduce to its proper limits the volume of blood supplied to the heart walls. Organic changes must necessarily inhibit this function. That the coronaries are the seat of such lesions in this disease is familiar to every one. Huchard's[81] summary of 145 autopsies shows that in 68 instances there were lesions of both coronaries, and in the 128 of the total in which the presence of such lesions is specified, 121 were atheromatous. That in the remaining few, lesions capable of interfering with the nervous functions of the vessels are present, though not advanced sufficiently to be discernible, is probable. Osler[82] states that "anatomically it has been shown that lesions of the coronaries are almost invariably present."

This involves the necessity of showing that angina pectoris can occur when the coronaries are patent. Jenner,[83] for example, to whom we owe the discovery that these vessels are diseased in this disease, did not find obstruction in the cases he examined post-mortem; in the one "the coronary appeared thickened;" in the other, the vessel itself was "discovered to be a kind of firm, fleshy tube." Osler,[84] who quotes these observations, states that the coronary arteries of John Hunter, who died of angina pectoris, "were found to be converted into open bony tubes." In a case reported by Beverley Robinson[85] in which there was "terrific suffering," the coronaries, "although uniformily affected by arteriosclerosis, were of large caliber and patent throughout, except near the end of a small branch," a fact which shows that the case "was not due" to "localized sclerosis and obstruction to the lumen of the coronary arteries." Other examples of this kind are available in literature.

Conversely, the coronaries may be diseased, narrowed, obstructed and even destroyed in part without giving rise to angina pectoris. Auscher[86] found the coronaries almost occluded by atheromatous plaques, in numerous autopsies of old people who had never suffered from the disease, and he refers to Pilliet, who confirmed his observations, and found a large number of obstructed arteries which had never caused angina. Tison[87] reported two instances in which, though the coronaries were atheromatous, calcified and rigid, with great narrowing of lumen, being scarcely permeable in one case, angina had never appeared. Osler[88] refers to a case in which, although no sign of the disease had occurred, the left coronary was "almost obliterated, only a pin-point channel remaining," while the main division of the right artery "was converted into a fibroid cord."

Much confusion prevails, in my opinion, owing to erroneous interpretation of post-mortem findings. That various substances found in the non-fluid state in the tissues after death are mobile fluids during life is shown by the post-mortem formation of myosin. That the coronaries and their branches are often found obstructed by substances which during life freely circulated through them is therefore probable. This relegates to fibrosis and calcareous degeneration the main rôle as sources of obstruction, and we have just seen that they may exist without causing the disease. That life can continue even to old age not-

[80] *Cf.* this volume, p. 1201.
[81] Huchard: *Loc. cit.*
[82] Osler: N. Y. Med. Jour., Aug. 8, 22, 29; Sept. 5, 12, 26; Nov. 7; Dec. 12, 1896.
[83] Jenner: Cited by Baron: "Life of Edward Jenner," 1827.
[84] Osler: *Loc. cit.*
[85] Beverley Robinson: Med. Record, Dec. 20, 1902.
[86] Auscher: Bull. de la Soc. anat., vol. lxvi, p. 545, 1891.
[87] Tison: *Ibid.*, vol. lxvii, p. 401, 1892.
[88] Osler: N. Y. Med. Jour., Aug. 22, 1896.

withstanding, shows that the remaining vessels, though strained perhaps, suffice to nourish the heart-wall. It is only when, as I interpret the process, *their* walls become diseased sufficiently to respond no longer to the controlling impulses of their vasomotor sympathetic nerves, and the blood can without restraint flood the myocardium, that the anginous attacks can occur.

When from any cause *general* vasoconstriction occurs and the blood-pressure in the body at large is raised beyond a certain limit, an unusual volume of arterial blood—varying in each case with the degree of control to which the coronaries are still subjected—is forced into the myocardium.* The myocardial vessels being thus inordinately dilated, and the *vis a tergo* motion of the blood being unusually violent, a forceful and mechanical hyperæmia of the nervi nervorum and neurilemma of the multitude of nerves and ganglia which the heart-walls contain is caused.* This evokes the primary stage of neuritis in the cardiac structures,* and, therefore, local neuralgia.

Nothnagel, as is well known, ascribed a form of angina which he termed "Angina Pectoris Vaso-motoria" to vasomotor spasm, and the pain to the sudden increase of blood-pressure and strain imposed upon the heart. Suggestive in the present connection is his observation that the increase of vascular tension *preceded* the attack. William Russell[89] reached a similar conclusion. In the four cases reported the pulse invariably indicated the advent of an attack by becoming hypertonic; and relaxation took place when the attack ceased. In a case reported by Dodd[90] sphygmographic tracings taken by Lauder Brunton showed a high blood-pressure during the height of the attack and a lowered pressure during the inhalation of amyl nitrite, which, as first shown by Brunton, relieves the attack. These observations are confirmed by those of Fraenkel,[91] who states that, although the pulse may be but slightly or not at all affected, the blood-pressure is always raised; this author refers to Pal as having also found the blood-pressure elevated during the paroxysm. G. A. Gibson[92] ascertained that the rise was very marked, varying from 160 mm. to 170 mm. Hg.

The cardiac pain, as I interpret it in the text, is readily accounted for by the innervation of the heart-walls. Osler, referring to Berkeley's[93] histological study of the myocardium, states that "everywhere throughout the organ—in the tissue beneath the endocardium and pericardium, through the muscle substance and about the blood-vessels—the nerves are in extraordinary profusion," including *sensory* nerve-endings demonstrated in the *arteries* by Thoma and in the connective tissues by Smirnow.[94] True, as Osler says, "the most extensive lesions, inflammatory, degenerative and neoplastic, may not excite a single painful sensation. Pericarditis of the most intense grade, with deep involvement of the myocardium, may give not the slightest indication of its existence." This only proves, however, that an additional factor is necessary in this

* *Author's conclusion.*

[89] William Russell: Brit. Med. Jour., Feb. 10, 1906.
[90] Dodd: *Ibid.*, Feb. 15, 1896.
[91] Fraenkel: Deut. med. Woch., Bd. xxxi, S. 569, 1905.
[92] G. A. Gibson: vol. xxviii, p. 52, 1905.
[93] Berkeley: Johns Hopkins Hosp. Reports, vol. iv, p. 112, 1894.
[94] Smirnow: Anat. Anzeiger, Bd. x, S. 737, 1894-95.

connection: that which I have shown to be the source of *neuralgic pain* elsewhere, *i.e.*, hyperæmia of the nerve-sheath itself. Indeed, Lancereaux and Peter[95] found in several cases distinct evidence of neuritis in the nerves of the cardiac plexus, a fact confirmed by Gilbert and Garnier.[96] Laënnec, Hope,[97] Trousseau[98] and other authorities regarded angina pectoris as a neuralgia.

When the coronaries are sufficiently diseased to render the development of an attack possible, the presence in the blood of any substance capable of stimulating the vasomotor center sufficiently can provoke an attack.* Normal physiological wastes, such as those that follow the ingestion of food, suffice, in advanced cases, to cause a paroxysm, especially when the kidneys are at all diseased. In the majority of cases, however, there is a history of gout, syphilis, influenza, malaria, scurvy or other disease of a debilitating character, and which, therefore, entail, especially after middle-life, hypocatabolism and its consequences, the formation of toxic wastes and acidosis. These conditions tend not only to cause an elevation of the blood-pressure, but sudden exacerbations of the same after dietetic indiscretions, exposure, etc. Worry, grief, shock, excessive drinking, smoking, venery, etc., also prepare the ground for angina pectoris by debilitating the organism and slowing all metabolic processes.* Fits of anger and overexertion, by suddenly causing the appearance in the blood of an unusual proportion of waste-products, likewise cause a sudden rise of blood-pressure* which, in fact, may cause instant death. In some cases the general vasomotor center finally becomes oversensitive, when the least physical effort, palpation, etc., will provoke a paroxysm.

Gilbert and Garnier[99] contend that all cases of angina pectoris are of toxic origin due to uræmia. Zilgren[100] observed four cases following a febrile tonsillitis, and ascribes the disease to toxic materials derived from the tonsils. Curtin and Watson,[101] for example, observed a large number of cases after an epidemic of the last-named disease. Gélineau[102] observed many cases among sailors previously debilitated by scurvy. Excitement such as that of quarreling also provokes a rise of the blood-pressure, as shown by a case reported by Heineman.[103] Russell,[104] moreover, emphasizes the fact that in arteriosclerosis the vessels "are prone

* *Author's conclusion.*

[95] Lancereaux and Peter: cited by Knott: Dublin Jour. of Med. Sci., May, Sept., 1897.
[96] Gilbert and Garnier: Presse méd., vol. vii, p. 263, 1900.
[97] Hope: "Treatise on Dis. of the Heart." third edition, Lond., 1839.
[98] Trousseau: "Cours de méd. clinique," Paris.
[99] Gilbert and Garnier: *Loc. cit.*
[100] Zilgren: Rev. méd de l'Est., vol. xxix, p. 613, 1897.
[101] Curtin and Watson: Inter. Med. Mag., Jan., 1893.
[102] Gélineau: Gaz. des hôpitaux, vol. xxxv, pp. 454, 466, 478, 1862.
[103] Heineman: Med. Record, Dec. 12, 1896.
[104] Russell: *Loc. cit.*

to become hypertonic from causes which are commonly regarded as trifling," and cites Pal's opinion to the same effect. Russell, moreover, states that "hypersensitiveness of the vasomotor center will explain what has long been recognized—that paroxysms of angina have as their main determining cause physical effort, mental emotion, or digestive disturbances"—a very judicious conclusion, but applicable only, as I specify in the text, to certain advanced cases. As I interpret this question, the vasomotor center is normal, as a rule, and only becomes hypersensitive when the blood contains substances which unduly stimulate it during a prolonged period.

The excruciating retrosternal pain is not due to the neural hyperæmia of the myocardium, but to forcible expansion of the aorta immediately above the heart.* This portion of the vascular system is not only subjected to the stress which the resistance of the abnormally constricted arteries of the entire body imposes upon it, but also to unusually powerful contractions of the cardiac muscle, whose activity is greatly enhanced by excessive volume of arterial blood circulating through it.*

Osler[105] says in this connection: "Baumes ranked the disease as a retrosternal neuralgia (sternalgia). Laënnec gave it his strong support and held that either the pneumogastric or sympathetic division of the cardiac nerves might be implicated, and with either of them the brachial plexus." Corrigan, Romberg, Bamberger and others held the same opinion. Allbutt,[106] recalling that the most acute pain is retrosternal, above the heart and on a level with the aorta, and, moreover, that aortitis gives rise to pain which resembles that of angina pectoris, considers the aorta as the only seat of pain. That his conclusion is warranted, in so far however as to the aorta being *a* seat of pain, is suggested by the self-evident explanation of it that my conception of the morbid process, as a whole, affords.

Treatment.—Measures which Arrest the Paroxysms.—The paroxysm being due to the presence of an excess of blood in the myocardium and violent expansion of the aorta,* our aim should be to deplete these structures. *Amyl nitrite,* by causing dilation of all arterioles of the body, and, in large doses, depression of the vasomotor center besides, and therefore retrocession of the blood in the great splanchnic area, fulfills this object.* The volume of blood being reduced in the cardiac area, the myocardiac and aortic tension cease,* and the pain likewise, almost at once when a few drops of this drug are inhaled. *Nitroglycerin* acts in the same way, though not so promptly, but its effects are much more lasting, the blood-vessels remaining dilated half-an-hour or hour, or longer after an injection of

* *Author's conclusion.*
[105] Osler: *Loc. cit.*
[106] Allbutt: Phila. Med. Jour., June 16, 23, 30, 1900.

$^1/_{100}$ grain (0.00065 gm.). This may be repeated as required to sustain the effect. When these agents cannot be readily obtained, two teaspoonfuls of *sweet spirit of nitre* in a wineglassful of water will afford relief while they are being procured.

The suffering may also be controlled with drugs capable of dulling sensibility of the heart-muscle, by causing constriction of its arterioles—including those that supply nerve elements—through their stimulating influence on the sympathetic center.* *Morphine,* $^1/_4$ grain (0.016 gm.) administered hypodermically, is very efficacious in this connection, when the lesions of the coronaries and its branches are not too far advanced, or the arterial tension in the cardiac area is not too great. The best results are obtained by giving it immediately after the amyl nitrite inhalations.* The effects of both drugs are thus insured and prolonged.*

This is accounted for by the fact that the drugs aid each other: amyl nitrite depletes the cardiac area, while morphine contracts the arterioles.* Much the same effect is produced by Waugh's method.[107] This clinician gives a granule of $^1/_{250}$ grain (0.00026 gm.) of glonoin every minute until relief ensues and the face flushes; he then deepens and prolongs the effect by giving atropine—which also contracts the arterioles*—$^1/_{250}$ grain (0.00026 gm.) every ten minutes till the mouth begins to become dry. It has often been noticed that morphine even in very large doses fails to produce any effect; the cause of this is self-evident when centrifugal distension of the cardiac and aortic vessels—in accord with my interpretation—is considered as the underlying cause of the pain.

Measures which Prevent the Paroxysms.—The continuous use of morphine, belladonna, the coal-tar products and other agents which stimulate the sympathetic center is not advisable. This center rapidly weakens under prolonged stimulation and the patient's general condition is aggravated.* Agents which depress the vasomotor center are preferable, their benumbing effect on the latter preventing the sudden elevations of the blood-pressure which evoke the accesses.* *Sodium bromide,* 20 grains (1.3 gms.), alternating with *chloral hydrate,* 15 grains (1 gm.), or tincture of *veratrum viride,* 15 minims (1905 U. S. P.) (0.9 c.c.) given once daily, repeated if need be, efficiently protects the patient against attacks.* Nitroglycerin may be given in $^1/_{100}$ grain (0.00065 gm.) doses three times daily. *Sodium nitrite,* 2 to 5 grains (0.13 to 0.3 gm.), may be used as

* *Author's conclusion.*
[107] Waugh: Therap. Gaz., Nov. 15, 1903.

a substitute when nitroglycerin begins to lose its effect. *Erythrol tetranitrate* in 1-grain (0.065 gm.) doses four times daily has been highly recommended. All these agents are vasomotor depressants.

Nitroglycerin is a useful agent in this connection. William Murrell[108] states that the best results are obtained when the spirits of glonoin are used. His preferred formula is:—

℞ Spiritus Glonoini (B. P.),
Spiritus Chloroformi, aa, ½ drachm (2 gms.).
Tinct. Capsici, 1 drachm (4 gms.).
Aq. menth. pip., ad 1 ounce (30 gm.).

A teaspoonful every four hours, an extra dose being taken immediately on the onset of the attack.

He advises that this be a stock bottle from which should be replenished three or four small bottles containing a drachm each, which can be carried in the vest-pocket for immediate use when needed. Erythrol tetranitrate has been recommended by J. B. Bradbury,[109] Adam,[110] Boughton Addy[111] and others. Huchard[112] deems it capable of maintaining continuously the vascular tension close to its physiological limits.

The attacks may be reduced and even prevented by *dietetic measures,* the most important of which is to limit the amount of food taken and to abstain from the use of meat, the catabolic products of which are especially active as vasomotor stimulants.* Late suppers are particularly harmful, and even dangerous. A *milk diet* of two weeks' duration, aided by one of the nitrites, is very efficacious, even in severe cases. The limited diet is sufficient in some instances to avert paroxysms, provided worry, violent exertion, and other exciting causes can be avoided.

Huchard[113] states that angina with endocarditis is relieved by an exclusive milk diet and theobromin for two weeks, then one week each month with sodium iodide. During the rest of the month a restricted diet is allowed. Osler regards the diet in many cases as "the central point in the treatment." Russell emphasizes the close relationship between an injudicious diet and a high vascular tension and adduces cases proving this contention.

The progress of the disease may often be stayed by the use of *potassium or sodium iodide,* 5 grains (0.3 gm.), gradually increased to 10 grains (0.6 gm.) after meals, after the patient has been on the restricted diet two or three weeks. *Thyroid gland,* 1 grain (0.06 gm.) three times daily, is also useful.*

* *Author's conclusion.*
[108] William Murrell: Med. Brief, May, 1897.
[109] J. B. Bradbury: Brit. Med. Jour., Apr. 10, 1897.
[110] Adam: *Ibid.*, Feb. 12, 1898.
[111] Boughton Addy: *Ibid.*, May 6, 1899.
[112] Huchard: Revue de thérap. méd.-chir., vol. lxx, p. 433, 1903.
[113] Huchard: Jour. des praticiens, Feb. 23, 1901.

By stimulating the adrenal center, catabolism is enhanced and toxic wastes do not form. The nutrition of the arteries being gradually improved, the primary morbid process, arterial degeneration, is antagonized. An important feature of the treatment is to avoid constipation and intestinal autointoxication. An occasional *saline purgative* suffices for this purpose.

Strychnine and *digitalis* are casually mentioned by some authors. Strychnine, being a vasomotor excitant, predisposes to attacks, while digitalis by enhancing the vigor of the heart's contractions may aggravate the attacks—a fact sustained by clinical observation. I regard these drugs as harmful as long as paroxysms are likely to occur.

CEREBRAL HÆMORRHAGE.

SYNONYMS.—*Apoplexy; Cerebral Apoplexy.*

Definition.—An effusion of blood in the brain due, in the majority of (idiopathic) cases, to rupture of an atheromatous artery, and the exciting cause of which is an unusually high blood-pressure. The pathogenesis is that of arteriosclerosis, including the rôle of the adrenal system in this disease.*

Symptoms.—*Prodromal symptoms* occasionally occur: headache or a sensation of fullness in the head, vertigo, tinnitus, excitability, abnormal sensations on one side of the body, especially the extremities, such as tingling, numbness and choreic movements.

The *apoplectic stroke* itself may be preceded by slight vertigo, slight aphasia and unilateral weakness, and paralysis of a leg or arm; but often without any such symptoms the patient drops suddenly, and, occasionally, after a few convulsive movements, becomes comatose. The face is dusky and even cyanotic, though sometimes pale, the pulse slow and relatively strong, full and tense; the breathing slow and stertorous, and often of the Cheyne-Stokes type, the cheeks being blown out on one side and the lips being flabby, owing to paralysis of the muscles of these structures. In ingravescent apoplexy the symptoms come on gradually, in keeping with the slow progress of the effusion.

In some—usually very severe—cases the face becomes very

* *Author's definition.*

pale or livid, the pulse and heart beat feebly, all muscles relax, and the patient may die after being comatose a short time, or he may remain unconscious some time, recover somewhat, and die in the midst of a second seizure; or again, he may improve and follow the course of a less severe case.

The peripheral effects of the cerebral lesion then become manifest, one side of the body, including the facial and ocular muscles (the eyes being turned toward the side of the lesion), being as though paralyzed. The cutaneous reflexes may be elicited by vigorous stimulation, pinching, pricking, etc., but, as a rule, all the reflexes are abolished at first on the affected side, and often on the normal side as well. The pupils are often unequal in size, contracted at times and dilated at others, and respond slowly or not at all to light. The feces and urine are usually passed involuntarily. There is often polyuria, the urine often containing sugar and albumin.

The period of *reaction* is initiated by fever, the temperature, one or two degrees F. below normal after the onset of attack, now rising to 101° F. (38.3° C.), or even to 103° F. (39.4° C.). After a period varying from a few hours to several days, the patient recovers from the coma, more or less dazed, restless or even delirious, especially if the fever is marked.

The paralyzed muscles may now become temporarily rigid and even spastic, but ultimately the loss of power again becomes manifest, affecting all the muscles of one side, though differences in degree of paralysis exist between the arm and leg, or between the face and extremities,—the leg escaping, for instance, while both the face and arm are paralyzed, etc. The cheek and lips of the corresponding side remain relaxed, as they were during the comatose period; the tongue, when protruded, is pointed toward the paralyzed side, and the loss of function of one-half of its muscles causes speech to be thick and almost unintelligible. The sphincter muscles may preserve their activity, however, at least for a time, and the reflexes, abolished at first on both sides, may now return, and also, after some time, on the paralyzed side, where they ultimately become exaggerated, though the electric reactions show no change.

The period of *resolution* begins when the paralytic phenomena show improvement, especially in the muscles of the leg.

As it is the extensors which recover their contractility, the limb remains stiff, and when the patient is able to walk, the leg is swung around in a semicircle at each step, the toes scraping the ground. This gives the patient the characteristic gait. It is regarded as an unfavorable omen when the arm is the first to improve. What improvement occurs in this extremity is in the flexors and pronators, the arm being thus drawn close to the body with the wrist flexed, the forearm semi-pronated and the fingers closed over the flexed thumb.

An important feature of this stage is that passive motion is readily performed. The paralyzed muscles are structurally normal, but being of the voluntary type they are not used and, failing to be nourished, become rigid, tend to contract and to remain fixed in this condition—the "contractures" so commonly observed in this disease. When this has occurred passive motion is no longer possible. Symptoms due to impaired nutrition are often discernible in other structures, the skin of the arms, for example, which may become red and lustrous and cold. The readiness with which extensive sloughing occurs on the paralyzed side in bed-ridden patients also points to impairment of the nutritional functions. Complete paralysis, even of the arm, does not always occur, however; after a few months in some cases, the use of the fingers, hand, forearm and arm is gradually and progressively regained.

The term "resolution" I apply to this period seems irrelevant when the frequency with which paralysis follows a stroke of cerebral hæmorrhage is taken into account. Yet, in the light of my views, as will be shown under "Treatment," such an untoward result is probably avoidable, and such being the case, "resolution" becomes applicable.

The symptomatology of the stroke itself varies considerably as to intensity in different cases, and the ultimate result corresponds, in a measure, with the severity of the primary phenomena. Among the *favorable signs* are: a brief duration of the coma; motion, even though slight, of the paralyzed limb during the first twenty-four hours; a moderate rise of temperature when reaction occurs (102° F., 38.9° C.), and restricted paralysis, brachi-crural, facio-brachial, etc. The *unfavorable signs* are: prolongation of the coma beyond twenty-four hours; marked hypothermia; initial convulsions; profuse sweating; cyanosis; conjugate deviation of the eyes; a steady rise of the