internal antipyretic or analgesic drugs, represented by ether, alcohol, chloroform, morphin, nicotin, antipyrin, etc., are true protoplasmic poisons, diminishing nerve sensibility, metabolic and secretory activity, and lessening all vital resistance to toxic agents in proportion to the quantity used, yet they continue not only to be used freely by a large proportion of the profession in the treatment of a great variety of diseases, and still more freely by the non-professional public.

EPIDEMICS OF HEART FAILURE.

The active ingredients in most of the "headache powders" and other popular remedies for migraine, neuralgia, insomnia, and general nervousness, belong to the coal-tar group of antipyretics. The same group of remedies were given freely by perhaps a majority of physicians in the treatment of influenza or la grippe, as it has prevailed since the epidemic of 1889-90. And during no previous epidemic of that disease has there been so great an increase of deaths from pneumonia, bronchitis, and so-called "heart failures," nor were they followed by so many severe and protracted sequelæ, characterized by a persistent sense of weariness, loss of muscular strength and nervous energy, frequent neuralgic and rheumatic pains, with impairments of the cardiac, respiratory, and digestive functions and unrefreshing sleep. A very large proportion of such patients readily refer the origin of their troubles to a severe attack of la grippe, often from one to three years previously, during which they took liberal doses of both anesthetic and antipyretic drugs, and have been taking more or less of the same class of remedies to relieve their pains, lessen nervousness, and encourage sleep at night ever since. Thereby they palliate, for a few hours after each dose, their pains and sense of exhaustion, and at the same time perpetuate the impairments of cardiac, respiratory, metabolic and protoplasmic activity, until many of them become permanent invalids, or die suddenly from so-called heart failure.

RESULTS OF COAL-TAR PRODUCTS.

During the last fifteen or twenty years the discovery of pathogenic bacteria and their ptomain or toxic products as causes of acute infections as well as some more chronic diseases, has induced nearly all recent writers on therapeutics and practice of medicine to represent heart failure as the chief source of danger instead of the excessive pyrexia of the preceding decade. Hence in their chapters, especially on pneumonia, influenza, diphtheria, typhoid and typhus fevers, etc., they emphasize the danger from this cause, and charge the practitioner to guard against it, even from the early stages of those diseases. Consequently, a group of remedies called cardiac tonics and stimulants is now occupying the same prominent position in practical therapeutics as the antipyretic group did two or three decades since; and, unfortunately, it is made to include some remedies more antagonistic in their effects than were any of those in the antipyretic group.

ANTAGONISTICS CLASSED AS SYNERGISTS.

As has been previously stated, so long as the profession were obliged to depend upon the objective and subjective phenomena furnished by the person taking any particular medicine, they called all such as rendered the action of the heart slower, cardiac and arterial sedatives; and such as increased the frequency of the cardiac systoles were ranked as stimulant and tonic. Accordingly, aconite, veratrum viride, strychnin, digitalis, strophanthus, cactus, etc., were all called cardiac and vascular

sedatives; while alcoholic liquors, ether, camphor, carbonate of ammonia, tea and coffee were ranked as direct cardiac and vascular stimulants and tonics. But when the modern processes of experimental therapeutics were applied to them it was soon demonstrated that the strychnin, digitalis, etc., so influenced the cardiac, respiratory and vasomotor nerve centers as to cause the cardiac action to be slower and stronger, and more tone or firmness of the arteries, with deeper inspirations; thereby showing them to be true cardiac and vasomotor tonics instead of sedatives. And as their action was not accompanied by coincident anesthetic influence on the cerebral hemispheres, they were readily transferred to the class of cardiac and vasomotor tonics with alcohol at its head. Consequently, we still find in nearly all recent practical works heart failure is set forth as the chief danger in the progress of all acute infectious diseases, to counteract which early resort should be had to cardiac and vasomotor tonics, of which we are uniformly assured that "alcohol, strychnin, digitalis and strophanthus are the most reliable." Thus placing two remedies at the head of the list, to be given alternately or even simultaneously to the same patients and for the same purposes, that have been demonstrated to be as near absolutely antagonistic in their influence on the cerebrospinal, vasomotor, metabolic and excretory functions as any other two remedies to be found in the materia medica. The one, alcohol, diminishing all the functions named, while by its anesthetic effect it renders the patients more quiet and less conscious of either weakness or pain; and the other, strychnin, increasing them, and thus leaving the physician to wonder why the patient tolerates so large an amount of medicines with so little apparent effect. Is it not fully time that such inconsistent medication should be avoided?

TRANSIENT MONOCULAR BLINDNESS.*

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Apart from the momentary blurring of vision from a tired ciliary muscle, or the partial blindness which accompanies migraine, or the periodic dimness of sight which is so frequent a precursor of glaucoma, transient loss of vision limited to one eye is very uncommon.

The attention of the writer was first called to this symptom some years ago by a patient, an elderly clergyman, who was brought to the office in a great state of perturbation by a friend, on account of the sudden loss of sight in the right eye. The blindness was complete for about fifteen minutes and then began to clear, so that by the time the office of the writer was reached, about half an hour after the commencement of the attack, sight was fully restored. A careful ophthalmoscopic examination was at once made, but failed to reveal any cause for the blindness, other than changes in the retinal vessels, which indicated a high degree of arterial sclerosis; thickening of the walls of the arteries and veins and too pronounced a light reflex upon them. The veins were slightly enlarged and pulsated upon the disc. Precisely similar changes were observed in the left eye. There was nothing to note externally beyond a pronounced degree of arcus senilis in both eyes. After the correction of a moderate amount of far-sighted astigmatism, the vision of both eyes was brought to normal.

* Read before the Section in Ophthalmology of the College of Physicians in Philadelphia

Upon questioning the patient it was ascertained that a similar attack of blindness had occurred in the same eye three weeks previously, the loss of sight at that time having persisted about ten minutes. Since there appeared to be no ground for alarm so far as the immediate safety of the eye was concerned, the fears of the patient that he would lose his sight were allayed and he was dismissed with the request that he see his physician at once for a thorough examination of his physical condition, as it occurred to the writer that the blindness was probably of a vascular origin. The patient was the subject of rheumatoid arthritis of marked degree and it was thought not unlikely that there had been changes in the blood vessels induced by this disease. Three days later he returned a second time, even more anxious than before, as he said that the blindness had returned for the third time and that it had persisted, though with intervals of remission, for five or six hours. An ophthalmoscopic examination was at once made with a negative result. His physician reported his kidneys and heart to be normal and his general condition excellent, considering the ravages of the rheumatoid arthritis, which had produced extensive deformities in the small joints. His diet was curtailed and he was placed upon active saline treatment. Two years have now elapsed without a recurrence of the blindness. To sum up briefly: all three attacks occurred in an interval of less than a month; the blindness always affected the right eye; the loss of sight was sudden and complete, the patient being unable to see a lighted match before the eye; the vision of the fellow eye was unaffected. Recovery of sight was rapid, beginning in the temporal field, and was preceded by the appearance of phosphenes. The attacks were not accompanied by headache, nausea or other general symptoms and so far as the patient knew were not precipitated by any unusual activity of mind or body.

Another case of the same nature, a lawyer, aged 30, came under the observation of the writer 7 years ago on account of twitching in the lids after prolonged near work. He had had headache but rarely and had never experienced any former difficulty with his eyes. Examination revealed fibrillary twitching in the orbicularis of both eyes, more marked on the left side, especially when that eye was made to fix. Vision and accommodation were normal in both eyes. At 5 meters esophoria equaled 2 degrees; at 40 cm. there was exophoria of 2; hyperphoria was absent. The eyegrounds were normal. Refraction under mydriasis showed a moderate degree of farsighted astigmatism in both eyes. Under a partial correction of this error the twitching subsided in large measure, but it was found that a full correction was necessary to cause its full disappearance.

About a year after he had worn the correction the patient returned, saying that he had noticed on two occasions that the lower half of objects became suddenly obscured. No headaches followed the loss of sight, though he thought he remembered seeing a play of lights before the eyes. He was unable to say whether both eyes were affected or not. A month later he had another attack in which the right eye became absolutely blind for fifteen minutes. There were no other motor or sensory symptoms, and headache and nausea were absent. Three months after this, after reading, he noticed a dark spot, the size of a fifty-cent piece, over the right eye, when he regarded the flame of a lamp. The spot for a time was directly in the center of vision and then became eccentric. After persisting half an hour, this phenomenon passed slowly away. One month later the upper half of

his right eye became totally blind for fifteen minutes. Vision was then undisturbed for six months when the lower half of the same eye became blind for five minutes. Nine months later the right eye became totally blind again, the loss of vision beginning in the lower part of the eye. In writing of this attack the patient expressed himself as follows: "I was standing on a street corner talking to Mr. M. when a dimness came over the lower part of the right eye. Thinking my glasses were fogged, I removed them for the purpose of cleaning them, when I found that the trouble was in the eye. The sight became rapidly dimmer, so that in a few moments I could hardly distinguish light with that eye. I was feeling perfectly well at the time, and after the vision cleared, which happened in a few moments, I resumed the conversation." A week later he had another attack of dimness in the same eye, the loss of sight being more marked in the upper nasal field. During the last three years he has had several other attacks of blindness in the right eye, the loss of vision usually beginning in the lower part of the eye.

As this patient lived at some distance from the city the writer has never had an opportunity of making an ophthalmoscopic examination during an attack, but in the interval the findings are negative. This patient, though young and vigorous, led a sedentary life, has had several attacks of rheumatic fever and came of a gouty family.

About 16 months ago the writer had the opportunity of studying a third case, that of a man aged 50 years, who consulted him on account of a sudden loss of sight in the right eye. He said that the eye was so blind that he could not see his hand even when it was held close to the eye, for at least one hour, and that during still another hour the fog was so thick that he could barely distinguish large objects. There was no headache or other accompanying symptoms. Since then he has had six other attacks, but not always in the same eye; the third and the last two occurring in the left. All of the attacks came on after taking a nap in the middle of the day. The second and third attacks lasted 30 minutes, and the last three but five minutes. The optic nerves in both eyes were somewhat grayer than is usual, but central and peripheral vision were unaffected. The retinal vessels showed no changes beyond a slight fulness and tortuosity of the veins. So far as could be ascertained, the patient was in perfect health.

In addition to these three, the writer remembers two other cases of a similar nature. One of these, a young woman who was epileptic, said that quite independent of her epileptic seizures she had had transient attacks of blindness in one eye which lasted from two to ten minutes. Loss of sight was sudden and complete; both eyes had been affected but never at the same time. No other symptom accompanied the blindness. Her epileptic attacks were without visual auræ.

In the remaining case, that of a young married woman twenty years old, the blindness, which affected the right eye only and lasted from five to ten minutes, had appeared several years previously and had recurred on an average of once a month. The patient had had several miscarriages and was said by her physician to suffer from uterine disorder. There seemed to be no connection between the loss of sight and menstruation. There was a large patch of choroiditis in her left eye, which looked as though it might have been of a vascular origin.

Though cases of this nature can not be rare, for some curious reason, except where the loss of vision has oc-

curred in connection with embolus and thrombosis of the central artery of the retina, transient attacks of blindness in one eye have escaped description in the textbooks, and have received study by but a few. It so happens, however, that these few have all been leaders in the ophthalmologic world, so that the observations which have been recorded are all instructive and comprehensive.

Loring in 1874 was the first to make any extensive communication upon the subject of transient attacks of blindness, although Alfred Graefe, Hedaeus, Rothmond, Secundi, and others had reported isolated cases, where this symptom had occurred in connection with excessive reduction in the diameter of the retinal arteries. Loring based his study upon the observation of five cases, in all of which symptoms previously ascribed to embolism of the central artery of the retina were present. This origin of the blindness, however, he disputed and concluded that, in the train of symptoms classed under the title of embolism, too much significance had been given to the stoppage of the circulation by the lodgment of a plug formed at a distance from the eye, while not enough stress had been laid upon the mechanical actions regulating the supply of the blood within the eye, as well as upon the condition of the walls of the vessels themselves and their contents. In explanation of the blindness, he says: "Might not the blindness be the final result of a series of processes which had been smouldering for some time and which had ended in a stoppage, or at least in a reduction of the circulation. This sudden shutting down of the circulation occurs in other parts of the body and why not in the eyes? A rapid transudation of serum from the capillaries and small vessels might so interfere with the fluidity of the blood as to render it unfit for circulation, or the stoppage might be brought about by some antagonistic condition of the walls of the vessels themselves. In a membrane so delicate as the retina, and one which the simplest experiment proves is so absolutely dependent upon the circulation for the performance of its functions, the slightest disturbance might seem to overthrow the equilibrium of the circulation and thus bring on, just as Donder's experiment does, almost instantaneous blindness."

A few years later (1879), Nettleship added largely to our knowledge of the subject by contributing two cases of repeated paroxysmal failure of sight, in connection with heart disease. He offered the hypothesis that the loss in vision was due to some permanent local disease of the ophthalmic artery or its branch to the retina in conjunction with a feeble stream of blood entering the eyes as a consequence of cardiac trouble.

In 1884 Priestley Smith reported a series of cases, which supported in the main the conclusions arrived at by Loring and Nettleship. The chief point made by him, however, was that permanent, as well as transient attacks of loss of sight from diminished blood supply to the retina, are usually of thrombotic origin and may occur in persons other than those who have organic disease of the heart. In support of this theory he reported a number of cases where he thought the blindness was due to disease of the sexual organs and explained the pathologic sequence in these by reflex disturbance of the circulation in the brain and retina through cardiac inhibition; great reduction and slowing of the blood stream in the retinal artery and finally thrombosis; this latter being strongly favored by anemia and feebleness of heart.

The next communication of importance upon the sub-

ject was not until 1897, when Wagenmann reported a case before the Congress at Heidelberg, of repeated attacks of loss of sight in one eye, which finally culminated in the eye becoming absolutely blind. He attributed the blindness in this case to a spasm of the central artery of the retina, and thought that this was secondary to arteriosclerosis, of which there was ophthalmoscopic evidence.

All of these authors, therefore, attribute the loss of sight to some disturbance in the vascular supply of the eye. Indeed, in the absence of any pathological findings in the fundus after attacks of blindness such as have been described, it is natural to impute the loss of vision to some spasmodic affection of the vessels, the spasm not being maintained long enough to cause any permanent change in the tissues.

In all of these cases, the blindness, though complete for a time and recurring in several instances over many years, was transient, and the vision, after being suppressed for a varying interval, returned. It would appear from this that such attacks of blindness are not to be dreaded, that they are not deleterious to the eye and that they have no significance other than the slight annoyance which they occasion at the time the vision is affected. The prognosis, therefore, should apparently be excellent. The surgeon, however, who would not give a guarded opinion in this class of cases would commit a grievous error, for it sometimes happens that the blindness which had before been transient becomes permanent, and as there is no way to differentiate the cases in which this unfortunate result occurs from those in which the loss in vision is only transient, it is wiser for the surgeon not to commit himself as to the final result. Of particular interest in this connection are two cases which have recently come to the attention of the writer. The first, J. L. W., aged 40, a manufacturer, consulted him on January 13, this year, on account of the sudden loss of sight in his right eye two days previously. The patient said that 10 years ago while sitting at the table eating, he suddenly became totally blind in both eyes; that this condition persisted for two or three minutes, vision slowly returning at the end of that time. His wife corroborated this statement and added that, with the exception of the disturbance in vision, the patient appeared perfectly well; his face was neither blanched nor suffused, nor did he complain of feeling faint or giddy. At varying intervals after this, he had spells of double vision, one object being higher than the other. The diplopia would last about five minutes, coming on without apparent cause, and was usually manifested when regarding distant objects. These attacks were not associated with nausea or other general symptoms and recurred every week or so. Eighteen months ago, these attacks ceased and he began to have spells of blindness in his right eye. These came on suddenly and lasted but a few minutes. At times the loss of sight was complete and at times but partial. There were 15 or 20 attacks in all. He was never conscious of headache, nausea or other unusual sensations at the time of the attack. The patient was a large, finely developed man of irreproachable habits, he had always been well, save for vague rheumatic pains in his back and shoulders some years previously. His bowels were regular and an examination of his urine showed his kidneys to be normal. His heart was also normal, though his family physician, Dr. U. G. Heil of West Philadelphia, who referred the case to me, stated that there was evidence of cardiac hypertrophy. About 10 years previously, at the

same time that he had noticed the first interference with his vision, he began to have curious smothering sensations in talking, as he expressed it, as though there were a stiffness of his jaws. This was always exaggerated by the embarrassment occasioned by talking to a number of people.

When inquiry was directed particularly to the loss of vision for which he consulted the writer, it was elicited that the blindness had been preceded by a few minutes of flickering, but that it had then become complete and so persisted for 6 or 8 hours, when it began steadily to improve, until it equaled, as it was ascertained at the examination, counting fingers in the upper part of the field at one-half meter's distance.

As was expected from the history, the ophthalmoscope revealed all the signs of a serious interruption in the circulation of the retina. The optic nerve was swollen and its edges obscured, particularly the upper temporal. The retinal arteries and veins were much reduced in size, particularly the superior branches and the veins in this region were twisted and cordlike. The retina was hazy and swollen above and in the papillomacular region. The lymphatics of the head of the nerve were full. As was indicated by the field blank, that portion of the retina supplied by the superior temporal and the superior macular branches of the retinal artery was blind. The left eye was normal, there being no unusual manifestation in the fundus, vision equaling 5/5.

A diagnosis of thrombosis, or perhaps merely of spasm of the central artery of the retina or of its superior branch, was made and the patient was placed in bed upon active treatment, with a view to reestablishing the circulation in the retina. Gentle but firm massage movements were made upon the globe, the bowels were freely opened and mercury and potassium iodid were administered in large doses. The vision improved gradually, so that at the end of a few days it equaled 5/40. After this treatment had been persisted in for three days and the vision was slowly improving, one day, shortly after eating, the patient again became suddenly and completely blind in his right eye for several minutes. This was due, in the opinion of the writer, to a spasm in the vessel, and indicated in all probability that the structural changes which had occurred in the retina were possibly due to that cause also, without a thrombosis having necessarily formed in the vessel. The patient has been out of bed now for several weeks, walking about and attending to his business; vision is slowly improving, but as is to be expected, the loss in the field still persists.

The second case occurred in the practice of Dr. Julian C. Dewey, to whom the writer is indebted for the report. In this instance the patient, a healthy man, aged 39, had had repeated attacks of blindness in the left eye since he was 8 years old, the loss in vision lasting from five to ten minutes. The attacks had happened so often, however, that the patient had become quite accustomed to them and their appearance had long ceased to occasion him any alarm, as from his past experience he judged that the dimness would soon pass away. A short time ago, however, he suffered another attack, apparently quite similar to the many which had preceded it, but this time, to his great alarm and surprise, the vision had failed to clear and the blindness persisted. He hastened to Dr. Dewey for relief, but without avail, as the ophthalmoscope, which up to this time had revealed nothing

abnormal, showed all the evidences of embolus of the central artery of the retina.

An observation of very great interest in connection with these cases, both from pathologic and physiologic standpoints, is one recently made by Leber, of a case of monocular blindness where there were all the ophthalmic signs of embolus but in which the microscope failed later to reveal any evidence of either embolism or thrombosis. Leber attributed the blindness and the pathologic findings to long-continued spasm pure and simple. The course of both of these cases, until the final attack came and vision was hopelessly lost, was precisely analogous to the apparently innocent group of transient blindness. It is evident, therefore, that the prognosis in all of these cases of transient blindness should be most guarded, as it would appear impossible to predict what the outcome of any one of them might be, whether the blindness be due to spasm and be always transient, whether it be due to a forming embolism which finally destroys the sight by shutting off the blood supply entirely, or whether the same fatal result is attained as in the case reported by Leber, by long-continued spasm, without microscopic change in the vessel walls.

In view of the uncertainty regarding the nature and outcome of these attacks, it is of the greatest importance to prevent a recurrence of them, by combating any tendency towards endarteritis, as it is probable that the spasm in the walls of the vessels is induced by such a process. It has long been a recognized fact that iridectomy, by causing a dilatation of the blood vessels, reduces intraocular pressure. This operation has accordingly been performed in a number of cases of transient monocular blindness with a view to preventing subsequent permanent blindness. Wagenmann would have it performed in every case of this nature. In view of the uncertainty regarding the cause and the course of these cases, it does not seem proper to the writer to subject an eye, which may remain permanently healthy, to an operation which in a certain proportion of instances, no matter how skilfully performed, renders the eye useless for visual purposes. He would, however, insist upon a treatment and a regimen to combat arterial sclerosis. At the time of the attack, the value of nitrate of amyl has been proven, and gentle but active massage of the eye should always be essayed.

PERFORATING WOUNDS OF THE EYEBALL AND SYMPATHETIC INFLAMMATION.*

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Penetrating wounds of the eyeball constitute a fair share of ophthalmic practice. Where mechanical industries expose workmen to accidents, these injuries may represent nearly 5 per cent. of eye patients. Injuries of this kind are always so serious that the surgeon necessarily feels a grave responsibility until the healing is complete. Every penetrating wound is to be considered from three aspects, viz.: 1, the mechanical consequences; 2, the presence or absence of a foreign body, and 3, the question of infection.

THE MECHANICAL CONSEQUENCES.

Eye injuries are peculiar in their immediate results, for nowhere else in the body are so many separate tissues of distinctive individuality crowded into so small a space. A wound which perforates the cornea allows

* Abstract of lectures delivered at the Northwestern University Medical School, with omission of the more elementary details.