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THE "gilded-cage" of ten to twenty bedrooms, with but a single small, ill-ventilated "water-closet," held many a martyr to constipation or its alternative of the mid-Victorian era: Grandma's nauseating brews, or the doctor's unrefined castor oil or calomel.

With the passing of heavy red flannel underwear, the treatment of constipation has also emerged from its early crudity. Out of the welter of professional opinion for the most satisfactory modern treatment of this ever prevalent condition, crystallized the Agarol idea—a mineral oil and agar emulsion suitable for every age period and in every pathologic condition where an intestinal evacuant is indicated.

Physicians are using Agarol extensively for the relief of acute constipation and for the treatment of habitual constipation. They know that its high viscosity, thoroughly emulsified mineral oil accomplishes exactly what it is intended to do—soften the intestinal contents, while the experimentally determined dose of phenolphthalein assures adequate peristaltic stimulation and thorough evacuation.

AGAROL

When writing advertisers please mention University of Western Ontario Medical Journal.
Sir William Osler*

By Wilder Penfield**

The post of Honorary President of this Society has, up to the present moment, been a source of great satisfaction to me. Now I am at a loss to know how I can bring new knowledge of their patron saint to the members of this Society, already steeped as you are in Osler lore. His distinguished biographers, Cushing, Reid, Abbot, Thayer and many others, have already heaped his shrine high with tributes, higher perhaps than the "Saint" himself would have liked, and, for you who did not know him, there is grave danger that fulsome praise may hide from you the simplicity and charm of the man. But I can at least add a little information from new sources, and, perhaps, give you a different point of view.

Contemporary descriptions of Osler by his own students are rare, perhaps because they were too close, at that time, to see him in perspective. Consequently, most descriptions have been in retrospect.

In the letters by J. B. MacCallum, scientist and gentle poet, who died all too soon after graduation from Johns Hopkins, references to the Professor of Medicine are only occasional.

He writes as follows: "I was called up in Osler's Clinic . . . They are the nicest things we go to, for Dr. Osler sits on the table and swings his feet, and asks you all sorts of questions you have never heard before. . . . Dr. Osler's clinics are splendid. It is so nice to hear him talk to the patients. He has a joke for everyone."

And again, when young MacCallum was Osler's patient himself he wrote: "You can't get anything out of him. He is always talking such a lot of nonsense."

From the unpublished letters of another medical student, written to his mother, I have culled more extended references to Osler, now in

*Address of the Honorary President at the Fourteenth Annual Banquet of the Osler Society of the University of Western Ontario, London, January 24th, 1941.

**Director of the Montreal Neurological Institute.

1If you have not this book, "SHORT YEARS," edited by Malloch, I recommend it strongly for a special place on your bedside table.
his Oxford period. Without alteration of diction, I shall read some of these to you. This student, although he then knew only a little of Osler's heroic past, was surprised to find him an ordinary man, like other men; and a week after his arrival he wrote from his rooms in College:

Oxford, January 1915. "When I look up at the seven volumes of Osler's Medicine on my shelf it makes me, mentally, worship him. It does not seem possible that he can be the same middle-aged man I saw last Sunday, who, with a room full of guests, spent most of his time in pretending to bandage up the leg of a young officer, to the glee of two little children.

"Sir William was a regular kid, but he said to me: 'Don't you go to the Front; you have got to use all your vacations in real work. I'm going to watch you and see that you don't go home any vacation,' so I guess I'm committed to vacations in Edinburgh and will see little work in France."

In the letters of the next eleven months there were recurring incidental references to Sir William which need not be quoted. In December he wrote: "Sir William had the students of his department out to his home one evening and he talked very interestingly about the origination of Physical Diagnosis and showed us some of his priceless collection of early manuscripts and writings of doctors; old Latin things, for Latin was the only written language of medicine for a long while."

Oxford, January. "Davison\textsuperscript{2} just came in and said that Osler had 'phoned him and asked if he and I would not like to go down to Cliveden with him tomorrow; it means cutting one lab., but of course I will go. He goes to Cliveden each week to inspect the big Canadian Hospital there."

And again in February:

"Sir William told the story of his life last night, at a meeting of the American Club, simply, with no affectation nor false modesty. He said he started with every opportunity, seventh in a missionary's family, with twins ahead. He took time for a 'gilt-edged' degree and for working too. When the Hopkins was being built, he was at the University of Pennsylvania Medical School. He said one morning Dr. Billings walked into his room and said: 'Osler, we are opening the Johns Hopkins in a month, will you go down and take charge of the School of Medicine with Welch?' Osler said: 'Will I? Yes.' 'All right, someone will write you, good morning.'

"When he was in England in 1904, and tired almost to death with the work and engagements of Baltimore, they offered him this job here (the Chair of Regius Professor of Medicine). So he cabled his

\textsuperscript{2}Wilburt C. Davison, then Oxford undergraduate, now Dean and organizer of the Faculty of Medicine, Duke University.
wife. Her answer was characteristic—it was 'Don't procrastinate; accept at once. Better to leave Baltimore in a ship than in a wooden box.' So he accepted.

"He said, at the end, that his rule had been to like and sympathize with everyone. That's his creed, I think. He is the least sentimental and the most helpful man I've ever seen—the most lovable. You may believe that he is stimulating to me, too, and is on something of a pedestal. If I were not so dumb, I should have the nerve to hope and dream I might follow in his footsteps."

On March 24th, while crossing the Channel for a second trip to a Red Cross Hospital in France, the student was wounded on a torpedoed vessel. He was returned to the Military Hospital, Dover, from where he wrote as follows:

"This is easily the best ward in the hospital. I am learning lots, lots. Bedside manner, I think I've discovered, is nothing but the effect of the doctor's personality. A young, handsome doctor left me hating him after three minutes of hurried examination. It was not that he did not know, but that he did not care about me, or my feelings . . .

"My! everyone is nice. Both Sir William and Lady Osler and their cousin have written and Sir William telephoned."

Dover, April. "Received my first bunch of flowers. The first ever. They came from Lady Osler. I can hardly understand all their kind attention. A letter came from him yesterday to tell me about the surgeon who is in charge of me, Mr. Linington. He says he seems to be a good man to judge from his directories, and he remembered an article by Linington in the Lancet and told me to ask the latter about it. So I did, and he seemed quite pleased and brought it for me to read. This morning, Mr. Linington said he had heard directly from Sir William."

This was his way of helping from behind the scenes. A month later, it was the student's unbelievably good fortune to find himself in the Osler home at Norham Gardens, from where he wrote on an Easter morning in April:

"It is good to be so near Sir William. He does not dislike anyone. He sees good and something to admire in everyone, and I've seen his face cloud up when someone repeated a bit of scandal or criticism. He is full of vigor and energy.

"Last night he came into my room about 10 o'clock, as he has each night, in the red smoking jacket. I showed him an X-ray photograph and simple photograph of one of my ten cases at Ris Orangis, which Dr. Blake had operated on. He said it was unique, and advised me to publish it!"
"Breakfast comes to me in bed. He forbids my getting up before. The silver and the little portions seem good after Dover. Soon Revere and Sir William both come in to see what they can do. Revere is a captain in the R.A.M.C. but is home on leave waiting his change into the artillery. After I am dressed, Lady Osler comes in to talk a little. Never before have I been waited on like this. If I enter a room, Lady Osler gets me a pillow, and someone else a footstool, etc., until I sit down quickly in a sort of shame.

"Much of the nice days I spend on the terrace overlooking the garden and Oxford Parks. I never heard such birds as here in England. It is like a great choir, the quality of whose voices is ever changing. I read Physiology, or, perhaps, one of the books Sir William has brought me, on the endocrine organs. One of them is in Italian, a great tome, but I look at the pictures and puzzle out a few words.

"Two little kiddies came in to see ‘William,’ as they call Sir William, the other day and, to amuse them, he took them up to a second storey porch which overlooks the garden and from there he threw water down on Lytle and Davison, who had come to see me. Then, when Lytle put up a lady’s umbrella, which lay there, he poured a whole pitcher of water full on him, while the kiddies screamed with delight.”

After this two weeks idyll, the student moved back into his Oxford lodgings, but the kindness that emanated from the Osler household did not cease. Revere came in after a day’s fishing on the Thames and left a trout for him.

The last letter from which we shall quote began as follows: "Let me tell you what Sir William has done now. He had Davison and me to tea on Thursday afternoon and then we went down to his office in the Museum. Here was a great collection of medical books and of his own reprints. The books were about to be sent to the University of Louvain. ‘Now,’ he said, ‘you boys had better take what books you like here, about 20 apiece, and take a set of reprints,’ and he went off with his springy step, waving his hand as he slammed the door to cut off our attempted thanks. We took off our coats and dove in, carrying off 40 and, later, splitting them in my room. I have a dandy two-volume surgery, etc., etc. . . . But the reprints are the best of all. You’ve no idea what that man has written on — almost every topic in medicine. And now he tells us to bring the reprints to tea this afternoon and he will send them away to have each set bound and titled.

“I shall never do it, but I’d like to get a first class in the final examinations because of what Sir William will think.”

* * * * *

In these youthful letters there is nothing very extraordinary, but they tell you why every medical man and student, who knew him, loved
him and resolved to emulate him. You must visualize Osler as a simple man who never made his juniors conscious of the fact that they were in the presence of greatness and, what is more important, I do not think he himself ever gave a thought to the length of his own shadow. He had too lively a sense of humor for that and, besides, he was much too busy following his own rule of life, which was “to like and sympathize with everyone else.”

The young physician who can make this Oslerian rule his own need not worry about having a proper “bedside manner.” Indeed, this term carries with it so strong a taint of insincerity that it would no longer be applicable to him. Patients are all too prone to impute knowledge even unto the least intelligent of our profession and, whether this be a curse or a blessing, it is a heritage that comes to us directly from the temples of Aesculapius, to cloak our ignorance. Alas, the cloak has to be larger now for there is more to be ignorant about! But for the lack of sympathy, there is no cloak.

Lord Tweedsmuir, in his book called “Memory Hold-the-Door,” makes an amusing addition to the concept of bedside manner when discussing Robert Louis Stevenson. He said he had felt a suspicion that there might be, in his fellow Scot, a “pose behind his optimism and masculinity—the pose of an heroic invalidism, a variant of the bedside manner, for,” he added, “there may be a bedside manner of the patient as well as of the doctor.”

In the summer of 1917 the medical student, who has been quoted above, had found his way to Paris. There he received the following letter from Lady Osler:

“You will, I know, grieve for us when you hear that Revere died August 30th from wounds. It is too horrible to take in, and yet we expected it. I prayed Sir William might be spared this. We know little yet. The first news came from Major Harvey Cushing, who was with him at the C.C.S., and that comforts us so much. I am bothering you—by asking you to do this for me—but know you will not mind. So many of Sir William’s friends are in France, and I know all will have the New York Herald (Paris edition), and so I am asking you to put this among the death notices:

‘Died of wounds received in Belgium, Edward Revere Osler, 2nd Lieut., Royal Field Artillery, aged 21. Son of Sir William Osler, Bt., Regius Professor of Medicine at Oxford, and of Lady Osler.’”

That the death of this only child and dear comrade was the greatest sorrow that life brought to Sir William seems obvious. But, although we know his nights were passed in agony, that house which had gained the name of “Open Arms,” an asylum which had continuously shut its
guests away from the worries and cares of war-time, did not now become a place of lamentation.

The week-end which followed has been described for me by Dr. Robert Osgood of Boston. When he learned of Revere’s death, he promptly proposed to recall his acceptance of an invitation to visit Oxford, but he was informed that both Sir William and Lady Osler would be “distressed and almost displeased” if he did not come. Therefore, with misgivings, Dr. Osgood carried on with the visit, which he describes as follows:

“Sir William met me on the Oxford platform, gay, debonair, with a flower in his button-hole, and, as we drove to Norham Gardens, was as scintillating, humorous and charming as he possibly could be, without a suggestion of any lurking sadness.

“Soon we dressed for dinner, at which there were perhaps half a dozen guests who were spending the week-end, including a scholar, whose name I have forgotten, connected with the British Museum, a Canadian lieutenant, who was just having his leave from his regiment in London, and myself. It was a very merry dinner party and Lady Osler seemed as completely in control of herself and her emotions as did Sir William.

“After dinner, when the gentlemen had gone upstairs to smoke in Sir William’s library, he would pull down a non-medical book from his shelves and ask the scholar from the Museum something about it and his opinion concerning it, and it would be quite evident in a few minutes that Sir William was very much the more conversant with this non-medical book. He would then touch on some medical subject and address me and I would, of course, scuttle as gracefully as I could beneath his feet. He would then turn to the Canadian lieutenant and discuss with him the size of Gertie Miller’s ankles (she was then the leading vaudeville star) and he had considerably more knowledge of their size and pulchritude than the young lieutenant.

“So the evening went. With the ladies he was again, of course, the brilliant leader of conversation. Sunday, Lady Osler went to church. There was another very considerable party at luncheon. In the afternoon twenty-five American aviators were in for tea with gaiety unconfined.

“It was almost more than one could bear, this apparent gaiety, this complete obscuration of his real feelings, because it was war-time and the sporting thing to do. Lady Osler entirely caught his spirit and talked and acted in complete harmony with his mood. I fancy efforts like this may have lost him to the world too early.”

* * * * *

Finally, for your pleasure and mine, I would not sit down without
allowing Osler to speak, though briefly, in his own words and at different stages of his development. His addresses were not extemporaneous. They were the fruit of labor and of revision with the pruning hook. He planned his incidental reading with a view to the collection of material far in advance of each address.

As the years passed Osler continued to be a clinical observer and a teacher without peer but not a research worker. His new studies came to lie in the field of literature and so, against a background of advancing scholarship, in each address he spun new "strands of arduous thought."

In his first year as a member of the faculty of McGill University, then aged 25, he delivered the valedictory address to the graduates in medicine and surgery. It is interesting to examine this first effort because it is so lacking in the grace and charm of his later writing. This may be illustrated by the following successive excerpts:

"Gentlemen of the Graduating Class: The pleasant duty devolves upon me of offering you ... congratulations on your present success. . . ."

He then gave them advice which I dare say he was taking for himself as well: "Let the spirit of our medical moralist, Sir Thomas Browne, whose Religio Medici I would commend to your perusal, actuate you."

There follow echoes from his early training, wise precepts such as the following:

"You may feel aggrieved," he said, "and think yourself wronged or slighted; instead of giving vent to your feelings on such occasions, restrain them and remember the injunction: 'If thy brother trespass against thee, go and tell him his fault between thee and him alone; if he shall hear thee, thou hast gained thy brother.'

"A word now on the Temperance question, which is becoming an all-important one in Canada for us as medical men. That alcohol is a medicine, and a valuable one, nobody not blinded by prejudice denies." . . .

"Example, gentlemen, is better than precept, and by becoming teetotallers yourselves you will neither injure your health nor damage your professional prospects."

3 During the last war the distinguished neurologist, Sir Henry Head, in a beautiful poem, wrote the following lines:

"Once like a spider, in his patterned web,
Based on immutable law,
Boldly I spun the strands of arduous thought
Now seeming naught,
Rent in the hurricane of war."
“In conclusion, gentlemen, let us hope that wherever you go you will maintain the good name of your Alma Mater, and add to the lustre which surrounds her. Bend all your energies to the attainment of proficiency in your calling; work while it is yet day, that when your night comes it may be said of you as of Gerard de Narbon, one of Shakespeare's Physicians: ‘He was in what he did profess, well found’.”

In form and content this might suggest the influence of his early teacher, “Father” Johnson, and also, no doubt, of his own father. Preacher, as well as teacher, he was and remained, but he later learned to tincture advice with humor and understanding in a manner not suggested by the lines just quoted. His ability as a speaker and writer was acquired after years of hard work.

Ten years later, after accepting a call to the Chair of Medicine in the University of Pennsylvania, Osler gave his introductory lecture in Philadelphia, a lecture which has never been published, but which Dr. William Francis kindly placed in my hands in the Osler Library, with permission to use it for the purpose of this address. I have copied some excerpts from Osler's longhand notes as follows:

“In the race, gentlemen, upon which you enter today, success or failure, as the case may be, will depend very much on the life which is now behind you. If you have been idle, and wasteful of your time at school, it will be hard to acquire industrious habits here. Bury the past and start afresh today with the firm resolve to waste not an hour of the short and precious time which is before you.” . . .

“Beautiful and enticing as is the study of Anatomy, we cannot see its full beauty until in Physiology we study the relations of structure to function.”

“Above everything, gentlemen, come to the study of the diagnosis of disease with all the modesty at your command. Positiveness and dogmatism are inevitable associates of superficial knowledge in medicine. We so long for certainty in this changing world, and the younger we are the more we seem to need it.”

“The motto of each of you as you undertake the examination and treatment of a case should be ‘put yourself in his place.’ Realize, so far as you can, the mental state of the patient, enter into his feelings . . . scan gently his faults. The kindly word, the cheerful greeting, the sympathetic look . . .”

“Bear away with you to your work the spirit of my text which I give you last on purpose:

“The knowledge which a man can use is the only real knowledge, the only knowledge that has life and growth in it and converts itself
into practical power. The rest hangs like dust about the brain or dries like raindrops off the stones.'"

Dr. Francis has pointed out that at the end of the address occurs this tentative final paragraph:

"The transplantation of a man, as of a tree, is a risky process and not always successful. Under the changed conditions a fresh growth does not inevitably take place. I trust, Sir, that in my case, if the flowers and fruit are not apparent, you can at any rate see 'the tender leaves of hope'."

This paragraph was struck out and below it his cousin, Miss Jeanette Osler (1839-1936), who was his devoted admirer and helpful critic, had written, probably years after the reading:

"I do not like this, you were beyond all they could hope for when you came, and to speak of 'tender leaves of hope,' in your case savours of the pride that apes humility, though I know nothing is farther from your nature, but everyone does not know you so well."

She followed this with a new version of the paragraph which she herself had evidently composed.

This lecture has fresh charm and enthusiasm. It had never been re-worked for publication, and is therefore the more interesting.

Four years later, Osler delivered the Valedictory Address at the University of Pennsylvania before taking his leave from that institution to accept the Chair of Medicine at Johns Hopkins. How different is this from his first valedictory address, in Montreal, in style, polish, humour.

"It is my duty," he began, "to say a few words of encouragement and to bid you, in the name of the Faculty, Godspeed on your journey. I could have the heart to spare you, poor care-worn survivors of a hard struggle, so 'lean and pale and leaden-eye' with study; and my tender mercy constrains me to consider but two of the score of elements which may make or mar your lives—which may contribute to your success, or help you in the days of failure.

"In the first place, in the physician or surgeon, no quality takes rank with imperturbability, and I propose for a few minutes to direct your attention to this essential bodily virtue. Imperturbability means coolness and presence of mind under all circumstances, calmness amid storm, clearness of judgment in moments of grave peril, immobility, impassiveness, or, to use an old and expressive word, phlegm. It is the quality which is most appreciated by the laity though often misunderstood by them."

"Cultivate, then gentlemen, such a judicious measure of obtuseness
as will enable you to meet the exigencies of practice with firmness and courage, without, at the same time, hardening 'the human heart by which we live.'

"In the second place, there is a mental equivalent to this bodily endowment which is as important in our pilgrimage as imperturbability. Let me recall to your minds an incident related of that best of men and wisest of rulers, Antoninus Pius, who, as he lay dying in his home at Lorium in Etruria, summoned up the philosophy of life in the watchword, Aequanimitas."

In the paragraph which now follows I seem to detect a slight savouring of rebuke, for the youthful professor had learned to know what it was to be patronized in the "city of brotherly love."

"While preaching to you a doctrine of equanimity, I am, myself, a castaway. Reeking not my own rede, I illustrate the inconsistency which so readily besets us. One might have thought that in the premier school of America, in this Civitas Hippocratica, with associations so dear to a lover of his profession, with colleagues so distinguished, and with students so considerate, one might have thought, I say, that the Hercules Pillars of a man's ambition had here been reached. But it has not been so ordained, and today I sever my connection with this University.

"Gentlemen: Farewell, and take with you into the struggle the watchword of the good old Roman—Aequanimitas."

* * * * *

This watchword he himself took with him through his life. He had it placed on his crest when he was created a baronet. Aequanimitas seems to me to be an essential quality in his own character that explains his behaviour. He did not strain for greater accomplishment than he was capable of and took all things "in his stride"—even the death of Revere, at which time, as we have seen, that principle so steadied him and his household.

Sir William Osler devoted his mind to Medical Education, to the Study of Clinical Problems and to the Lore of Medical History. In all these fields he excelled, and yet it is not altogether because of these qualities of the intellect that Osler Societies have sprung up all over the English-speaking world—chiefly composed of students or of young physicians. The unique quality of this man had to do with the "heart."

I would have you see him, to use the words of the above-quoted undergraduate, as "the least sentimental, the most helpful, most lovable" teacher of medicine. He belongs to medical students of all time, as Lincoln belongs to common men everywhere, a man who grew to be what he was by dint of hard work, and in whose footsteps any undergraduate may dare to "hope and dream" that he may follow.
Coma

By S. Roy Korey, '41

No diagnosis is more difficult and imperative than that of a patient in coma. There are a great many conditions which may induce coma, but to maintain clarity and coherence the writer has limited his discussion to the more important causes. An attempt has been made to familiarize the reader with the routine examination of a comatose patient and the outstanding characteristics of the underlying diseases.

Coma is the unconscious state of an individual. The depth of coma can be roughly graded by the patient's response to external stimuli. In profound coma, a patient may not react to the most painful and violent stimuli. Clinically, one considers a person to be comatose when he can be aroused with great difficulty or not at all and appears to be in a state of unnatural sleep. This sleep is sometimes interrupted by convulsions and often marked by slow, stertorous, or irregular breathing.

Classification—In attempting to classify the causes of coma we realize since consciousness is one of the products of brain function its loss necessarily implies central nervous system damage. Broadly speaking, coma can be considered a type of brain failure. From this standpoint, there appears to be an apparent link between the varied mechanisms that produce coma.

I.—Central Nervous System Disorders:

A—Head injuries
   1. concussion
   2. contusion
   3. extradural haemorrhage
   4. subdural haemorrhage

B—Vascular disturbances
   1. haemorrhages
      a—intracerebral and capsular
      b—pontine
      c—subarachnoid
   2. thrombosis
      a—arterial
      b—venous-sinus
   3. embolism
   4. hypertensive encephalopathy

C—Inflammations
   1. encephalitis
   2. meningitis
D—Epilepsy  
E—Hysteria  
F—General Paresis (G.P.I.)  
G—Intracranial tumours  
H—Benign syncope  
I—Extremes of temperature.

II.—METABOLIC (Chemical):  
A—Diabetic coma  
B—Hypoglycaemia  
C—Uraemia  
D—Addisonian crises  
E—Hepatic insufficiency  
F—Poisonings  
   1. morphine  
   2. CO  
   3. alcohol  
   4. atropine  
   5. barbiturates.

III.—CARDIO-VASCULAR:  
A—Hæmorrhage  
B—Stokes-Adams syndrome  
C—Carotid sinus syndrome  
D—Heart failure.

IV.—MISCELLANEOUS:  
A—Eclampsia  
B—Pneumonia.

No matter what the cause of the coma may be, a well-defined routine method of examination of the patient is required to cover all the salient features and give the investigator direction.

Investigation of a comatose patient—

HISTORY—usually from a second party—  
1. onset—sudden, gradual, associated signs e.g. convulsions  
2. previous definitely known diseases, habits and general health  
3. previous coma  
4. age.

EXAMINATION

General—1. estimated age  
   2. obvious trauma or/and hæmorrhage  
   3. convulsions and twitchings  
   4. depth of coma.

Head Injury—inspection for depressed bone, blood or cerebrospinal fluid from a wound or cranial orifice.
Skin—colour, temperature, state of hydration, injection marks, rashes. 
Eyes—subconjunctival haemorrhage, corneal reflex, size, equality, light 
reflex of pupils, conjugate deviation, ocular palsies. 

Odour of the breath. 
Respiration—rate, rhythm, type. 
Pulse—rate, rhythm, type. 
Temperature. 
Chest—routine examination; signs of pneumonia, particularly. 
Heart—size and sounds; signs of heart failure; blood pressure. 
Abdomen—distension; obvious veins; rigidity. 
Nervous system—ocular or/and facial deviations; position of uvula and 
tongue; tone and paralysis of extremities; superficial and deep 
reflexes; neck rigidity and Kernig’s sign. 

Ophthalmoscopic examination. 

SPECIAL INVESTIGATION 
Routine: a. urine—albumin, sugar, ketones. (In special cases, test for 
specific poisons.) 
b. blood—N.P.N., sugar, Wassermann, chlorides and CO₂ 
combining power when indicated, specific poisons. 
c. spinal fluid—including a Wassermann. 

b. Electrocardiogram. 
c. Gastric analysis for drugs and poisonings. 

DIAGNOSIS: The method employed here in differentiating the pos-
sible causes of coma consists in discussing each sub-group and comparing 
these sub-groups. Whenever possible, charts have been substituted 
for description. 

CENTRAL NERVOUS SYSTEM DISORDERS 

HEAD INJURIES. Head injuries produce unconsciousness by (1) 
destruction of brain tissue with haemorrhage or laceration; (2) functional impairment of the nervous 
system by ischaemia and increased intracranial pressure. Contusion belongs to the former group while 
extra and subdural haemorrhages are primarily of the latter type. The 
following points differentiate between these two groups. Contusions 
are more serious and irremediable usually with coma supervening 
immediately, whereas dural haemorrhages have a better prognosis and 
exhibit a variable lucid interval before consciousness is lost. 

Contusion exhibits at first the concussive syndrome which includes 
coma, shock, faint respirations and dilated non-reactive pupils, flaccid 
paralysis of the limbs, absence of superficial and deep reflexes. This 
concussive picture is generally present in any case of severe head injury 
and is non-specific. The diagnosis of contusion awaits the partial recov-
ery of the patient for only then localizing signs of focal brain destruction
are evident. The localizing signs are those of cortical irritation or destruction. The irritative phenomena, sometimes present from the start of the coma, are manifested by convulsions in a special group of muscles or generalized in extent but marked by a constant point of initiation. Damage to the caudate nucleus may cause hemiathetoid movements. Destruction of brain tissue results clinically in paresis or paralysis of muscle groups with signs of an upper motor neurone lesion. Neurological findings of an upper motor neurone lesion may take a day or more to supervene and consist of paralysis of the limb muscles, exaggerated deep and absent superficial reflexes, increased tonus or spasticity of an involved limb clonus and a positive Babinski or Hoffman. Damage to cranial nerves 3, 5, 7, 9, 12 and their nuclei are also of localizing value. Care should be taken not to suppose that there is organic damage to nerve elements when the real cause of the ensuing paralysis is functional impairment due to increased intracranial pressure. For this reason, as a localizing sign, paralysis of muscle groups supplied by the oculomotor nerve is more diagnostically important than complete annihilation of all the nerve's actions. The abducens nerve has purposely been omitted for it is so commonly only functionally impaired that it loses its focalizing significance. Hemianopias and aphasias are obvious when the patient resumes a semiconscious state and therefore are late manifestations.

Of all the factors mentioned above that compose the clinical state of contusion, persistent coma, early presence of neurological focalizing signs and an absent or only moderate rise in intracranial pressure are the general differentiating points from dural haemorrhages.

An extradural haemorrhage is classically suggested by concussion coma followed by a lucid interval of one to 48 hours and finally the signs of increased intracranial pressure and coma. The signs of increased intracranial pressure are: headache, vomiting, often projectile and accompanied by nausea, a slow pulse, a rise in blood pressure, and papilledema. The site of head injury in this case is usually the temporal region of the skull and the bleeding vessel is thought to be either the sphenoparietal sinus or the middle meningeal artery. Dilatation of the ipsilateral pupil due to pressure paralysis of the oculomotor nerve is invaluable in lateralizing the side of the haemorrhage. Signs of cortical interference are also used.

Subdural haemorrhage is slow and venous and characterized by the comparative insignificance of the precipitating trauma. The injury is commonly ignored by the patient. A diagnosis is suggested only after a latent interval of many weeks when deterioration of the individual's efficiency and personality, coupled with signs of local brain pressure and a rise of intracranial pressure, are significantly present.
A more acute form of subdural haemorrhage is differentiated from extradural by Foster Kennedy as follows:

"Acute" Subdural Haemorrhage
1. occurs at all ages
2. contre-coup injury
3. lucid interval is longer
4. more likely to be bilateral.

Extradural Haemorrhage
1. usually young adults, also middle-aged
2. injury ipsilateral to trauma
3. lucid interval is shorter
4. more likely to be unilateral.

Incidental to a head injury, there may be an immediate loss of consciousness that is supposedly due to subarachnoid and subpial haemorrhages in association with cerebral vasospasm. This is called concussion coma and must be regarded tentatively as a coma of more serious nature until the patient’s recovery and course of events prove it otherwise. Concussion coma is diagnosed by eliminating all of the other types of coma producing head injuries and by the individual’s rapid (24 hours) return to normal (Table I.).

<table>
<thead>
<tr>
<th>Onset of Coma</th>
<th>Contusion at once</th>
<th>Extravascular Haemorrhage at once</th>
<th>Subdural Haemorrhage rarely at time of accident. Frequently days or weeks later</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lucid interval</td>
<td>none—coma may lighten or increase; usually persists</td>
<td>usually present; short—hours</td>
<td>invariably present; days to weeks</td>
</tr>
<tr>
<td>Increased intracranial pressure</td>
<td>commonly no rise; if present only moderate</td>
<td>always present; reaches greatest height here</td>
<td>always present</td>
</tr>
<tr>
<td>Localizing neurological signs</td>
<td>definite and always present; relatively early</td>
<td>occasionally present; best is ipsilateral dilatation of pupil—occurs early</td>
<td>vague frequently; occurs late</td>
</tr>
</tbody>
</table>

VASCULAR ACCIDENTS—The various types of vascular accidents must be distinguished from each other and in turn from alcoholism, morphine, poisoning, uræmia, and epilepsy.

A “stroke” varies from a mild form with no loss of consciousness and variable paresis to a severe type characterized by coma, convulsions, either flaccid or spastic paralysis, aphasia and neurological signs of an upper motor neurone lesion. The causes of stroke are haemorrhage, thrombosis and embolism of an important artery. The accom-
panying table (Table II.) gives some idea as to the characteristic distinctions between these.

**TABLE II.**

<table>
<thead>
<tr>
<th></th>
<th><strong>Hæmorrhage</strong></th>
<th><strong>Thrombosis</strong></th>
<th><strong>Embolism</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>45-65 years</td>
<td>25-45 with Lues</td>
<td>Any age</td>
</tr>
<tr>
<td></td>
<td>45 years plus</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Condition existing</strong></td>
<td>Hypertension</td>
<td>Hypertension</td>
<td>Subacute Bacterial</td>
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<td></td>
<td></td>
<td>Lues</td>
<td>Endocarditis;</td>
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<td>Mitral Stenosis;</td>
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<td>Coronary Throm-</td>
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<td></td>
<td>bosis; Phlebitis;</td>
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<td></td>
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<td></td>
<td>Auricular Fibrilla-</td>
</tr>
<tr>
<td><strong>Onset</strong></td>
<td>sudden</td>
<td>sudden—or rapidly</td>
<td>sudden</td>
</tr>
<tr>
<td></td>
<td>progressive</td>
<td></td>
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<tr>
<td><strong>Coma</strong></td>
<td>very deep</td>
<td>none, mild, or deep</td>
<td>usually none, or</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>mild, rarely deep</td>
</tr>
<tr>
<td><strong>Vomiting</strong></td>
<td>common</td>
<td>unusual</td>
<td>unusual</td>
</tr>
<tr>
<td><strong>Blood pressure</strong></td>
<td>elevated</td>
<td>elevated, normal,</td>
<td>normal or elevated</td>
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<td></td>
<td></td>
<td>lowered</td>
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<tr>
<td><strong>Blood in C.S.F.</strong></td>
<td>present or none</td>
<td>present or none</td>
<td>rarely</td>
</tr>
</tbody>
</table>

It is far from easy, and may at times be impossible, to be dogmatic in establishing the cause of a stroke. The signs overlap each other considerably.

A person suddenly stricken unconscious by a cerebro-vascular accident may easily injure his head in the subsequent fall and lead the investigator into a false diagnosis of head injury. Differentiation of stroke from concussion should be made at once if possible. Initially, there is a definite similarity between a patient experiencing a stroke and one suffering concussion. Flaccidity of the muscles and non-reactivity of the reflexes are characteristic of both conditions at the beginning. Convulsions, cranial nerve palsies, hypertension and the onset of neurological signs suggest a stroke. In a cerebro-vascular accident, the neurological signs usually present themselves a few hours following the subsidences of the nervous diachisis and are progressive. If there be hyperreflexia and hypertonia from the start, intracerebral hæmorrhage has occurred and unfortunately broken into the lateral ventricle. The prognosis in this case is poor.

A vascular accident involving the internal capsule is the most frequent type. It is usually characterized by hemiplegia and occasionally by ipsilateral supranuclear paralysis of the 3rd, 7th, or 12th cranial nerves.

Weber's syndrome designates destruction of one pyramidal tract in the cerebral peduncle. It consists of complete hemiplegia and crossed paralysis of the muscles supplied by the oculomotor nerve.
Intrapontine haemorrhage manifests itself by pinpoint pupils, crossed facial paralysis either supranuclear or nuclear, possible interference with the 5th and 6th cranial nerves, respiratory difficulty, and occasionally hyperthermia.

Subarachnoid haemorrhage is evidenced by signs of meningeal irritation, i.e., severe headache, stiff neck, opisthotonus, positive Kernig and Brudzinski signs. Lumbar puncture reveals a grossly bloody cerebrospinal fluid.

Occasionally, following cerebro-vascular mischief, glycosuria is present. This is not persistent and ought not confuse the diagnosis.

**TABLE III.**

<table>
<thead>
<tr>
<th>Cause of Coma</th>
<th>Pulse (P)</th>
<th>Respiration (R)</th>
<th>Breath</th>
<th>Depth of Coma</th>
<th>Paralysis</th>
<th>C.R.</th>
<th>C.R.</th>
<th>Alcoho</th>
<th>Opium poisoning</th>
<th>Ureacin</th>
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</thead>
<tbody>
<tr>
<td>Stroke</td>
<td>P—full, pounding, rapid, or slow</td>
<td>R—strong, weak, or alcoholic, rapid</td>
<td>P—irregular, no odour</td>
<td>C.R.—present</td>
<td>cannot be roused</td>
<td>none</td>
<td>none</td>
<td>sweet opium, usually no rousable</td>
<td>none</td>
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Epilepsy (grand mal) is ushered in by a constant aura and is typified by a Jacksonian convulsion which abates in a few minutes, resolving into sleep or postepileptic states. The sterotyped sequence of events, the brevity of the attack, and the lack of paralytic residua distinguish epilepsy from cerebrovascular accidents.

VENOUS CEREBRAL ACCIDENTS—Sinus thrombosis is difficult to diagnose as such unless a pre-existing mastoiditis or marasmus is detected. Superior longitudinal sinus thrombosis may appear as typical apoplexy or may cause a sudden great rise in intracranial pressure, bilateral papilloedema, and paresis of both legs. Frequently there is no coma until late.

Thrombosis of a cavernous sinus causes unilateral exophthalmos, oedema of the eyelids, and dilatation of the veins of the scalp.

G. P. I.—In certain types of this disease, epileptiform convulsions and apoplexy may appear. The presence of an Argyll-Robertson pupil, optic atrophy, and a paretic colloidal gold curve are suggestive. The colloidal gold curve cannot be relied upon, unless unequivocally paretic, for central nervous system haemorrhage and trauma will produce misleadingly imitative curves. In a comatose patient, confusion of paresis and true stroke is certainly excusable for the signs are very similar.

BRAIN TUMOUR—This may suddenly cause coma due either to haemorrhage within itself and associated brain destruction or by a great increase in intracranial pressure. Haemorrhage into a tumour will usually resemble apoplexy at the onset. It is only when the patient recovers from coma that a final diagnosis may be made. Bilateral papilloedema and engorged retinal veins indicate increased intracranial pressure and occur much more often with tumour than with arterial accidents. Tumours of the midbrain, parieto-occipital region, and cerebellum are responsible for the greatest percentage of papilloedema. The Foster Kennedy syndrome, papilloedema contralateral and optic atrophy ipsilateral to the tumour, indicates a growth along the sphenoid ridge.

EXTREMES OF TEMPERATURE—The environment in which the patient is found offers the first and most important lead in diagnosis.

Heat stroke—the patient may be in a shocked condition with a subnormal temperature but not comatose or he may be unconscious, flushed, dehydrated with hyperthermia of a severe and oftentimes uncontrollable type.

Exposure to cold suppresses all the bodily functions and the patient lies inert with quiet, slow breathing, cold blanched skin, and slow, weak pulse.

The history of exposure and the characteristic appearance of the patient formulates the diagnosis.
INFLAMMATORY DISEASES OF CENTRAL NERVOUS SYSTEM CAUSING COMA—Encephalitis lethargica occurs usually in epidemics per se or associated with influenza. Its onset is variable, ranging from “grippe” to sudden apoplecticiform convulsions. The sequence of signs and symptoms are non-specific. The diagnosis is made by finding a lethargic or comatose patient, rather easily aroused, showing signs of visual disturbances, ocular palsies, upper motor neurone lesions, general muscular weakness with possible meningitic, neuritic, or irritative cortical phenomena. Differentiating this from meningitis is done most reliably by spinal fluid examination. The fairly constant signs of meningitis—fever, prostration, neck rigidity, opisthotonus, and positive Kernig and Brudzinski signs—are found only infrequently in encephalitis lethargica and so may safely confirm a diagnosis of meningitis.

Table IV. gives a short summary of the differential finding in the spinal fluid in the various meningitides, anterior poliomyelitis (which occasionally causes coma) and encephalitis lethargica.

SYNCOPE is not easy to differentiate from other conditions of a less benign nature. It is found more commonly in women, provoked by crowding, hunger or emotion. There are no convulsive phenomena nor characteristic neurological signs. Sequelae are usually absent. Laboratory investigation is negative and, in fact, rarely performed because the patient recovers so rapidly. The diagnosis is made by exclusion of other more serious conditions.

EPILEPSY AND HYSTERIA—Epilepsy frequently has prodromal symptoms, vague unrest, formications, or gain in weight some days previous to the convulsion. The attack is introduced by individually constant sensory, motor, or psychic aura followed by immediate loss of consciousness. The patient may emit a long expiratory cry and then fall to the ground in a state of tonic contraction. He becomes rigid, cyanosed, incontinent; may foam at the mouth; and then passes into a clonic spasmodic stage. On recovery from the fit which lasts one to two minutes, the patient falls asleep or rarely exhibits a postepileptic phenomenon like wandering about in temporary amnesia. The rigid sequence of events, the brevity of the convulsion and the normal blood pressure are excellent indications for a tentative diagnosis of epilepsy. Head injury occurring with the fall may confuse the investigator but further inspection postconvulsively will sustain one’s diagnosis.

Hysterical coma may simulate any other type with confusing results. The diagnosis is made by eliminating organic nervous disease through the absence of credible anatomical neurological findings and the general irregularity of the clinical picture. Examination reveals many previously unheard-of aberrations of the sensory and motor systems. The coma is not deep and the doctor’s suggestions frequently
TABLE IV.

<table>
<thead>
<tr>
<th>Pressure</th>
<th>Color</th>
<th>Number of Cells</th>
<th>Cell Types</th>
<th>Albumin or Globulin</th>
<th>Sugar</th>
<th>Chlorides</th>
<th>Organisms</th>
<th>Gold Colloidal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pyogenic Meningitis</td>
<td>++ + turbid to purulent</td>
<td>thousands</td>
<td>polymorphonuclear leucocytes</td>
<td>+++++ disappears 650-680 mgm. %</td>
<td>present</td>
<td>meningitic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Meningococcal Meningitis</td>
<td>++ + turbid to purulent</td>
<td>1-2000</td>
<td>polymorphonuclear leucocytes</td>
<td>+++ reduced 650-680 mgm. %</td>
<td>present</td>
<td>meningitic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T. B. Meningitis</td>
<td>++ clear with a pellicle</td>
<td>50-100</td>
<td>monocytes lymphocytes</td>
<td>++ reduced 550-610 mgm. %</td>
<td>Occurs in pellicle; guinea pig inoculation</td>
<td>meningitic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aseptic Meningitis</td>
<td>++ clear turbid purulent</td>
<td>10-200</td>
<td>polymorphonuclears then lymphocytes</td>
<td>++ normal</td>
<td>normal</td>
<td>normal</td>
<td>none</td>
<td>meningitic</td>
</tr>
<tr>
<td>Encephalitis Lethargica</td>
<td>+ clear</td>
<td>10-300</td>
<td>lymphocytes</td>
<td>+ increased normal</td>
<td>normal</td>
<td>none</td>
<td>paretic or tabetic</td>
<td></td>
</tr>
<tr>
<td>Anterior Poliomyelitis</td>
<td>normal clear hundreds</td>
<td>polymorphonuclears lymphocytes</td>
<td>+ + normal</td>
<td>moderate reduction</td>
<td>none</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
penetrates the patient's clouded consciousness with subsequent alteration of the clinical picture. Initially, hysteria may require differentiation from epilepsy.

<table>
<thead>
<tr>
<th>TABLE V.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>Cry</td>
</tr>
<tr>
<td>Fall</td>
</tr>
<tr>
<td>Sphincters</td>
</tr>
<tr>
<td>Tongue</td>
</tr>
<tr>
<td>Struggling</td>
</tr>
<tr>
<td>In presence of others</td>
</tr>
<tr>
<td>Duration</td>
</tr>
<tr>
<td>Sleep</td>
</tr>
</tbody>
</table>

**Metabolic Disorders**

This class of coma may be divided broadly into two rather well-defined groups: the endogenous including diabetic coma, hyperinsulinism, Addisonian crises, uræmia, hepatic insufficiency, and the exogenous made up of the intoxications. In the first division the onset of coma is on the whole more insidious and gradual with a well-established history of previous disease. (Hyperinsulinism onsets rapidly and is an exception.) Exogenous poisonings are characterized by a comparatively sudden appearance of coma in suspicious circumstances, e.g., in a closed garage with car motor running.

In attempting to differentiate the comas of diabetes, hyperinsulinism, and uræmia one is forced to conclude that the prodrome to the coma is the only period in which a clinical diagnosis can be made. The actual comas of these three conditions resemble each other so closely that, besides a few very rare characteristic signs mentioned below, we are dependent upon a laboratory diagnosis. Even here some equivocation in interpretation may exist. For example diabetic coma can be complicated by a prerenal azotæmia with clinical and laboratory indications of uræmia. Usually it is the laboratory data that is the final arbiter in these disorders.

The crises of Addison's disease are sometimes marked by coma. The preceding history of asthenia, nausea, vomiting, diarrhoea and the presence of a characteristic pigmentation, hypotension, and circulatory failure suggest the diagnosis. The prevailing coma is not profound.
TABLE VI.

<table>
<thead>
<tr>
<th>Precipitating Factors</th>
<th>Diabetic Coma</th>
<th>Hyperinsulinism</th>
<th>Uræmia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection; omitted meal or insulin injection</td>
<td>usually unheralded by any special event</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal Pain</td>
<td>+++</td>
<td>---</td>
<td>Less frequent—due to gastrœnteritis or strain of retching</td>
</tr>
<tr>
<td>Nausea and Vomiting</td>
<td>++</td>
<td>---</td>
<td>++</td>
</tr>
<tr>
<td>Hunger and Thirst</td>
<td>thirsty</td>
<td>hungry</td>
<td>thirsty</td>
</tr>
<tr>
<td>Respiration</td>
<td>acidotic (Kussmaul)</td>
<td>shallow</td>
<td>normal or acidotic</td>
</tr>
<tr>
<td>Odour of Breath</td>
<td>fruity and acetone</td>
<td>no odor</td>
<td>ammoniacal</td>
</tr>
<tr>
<td>Skin</td>
<td>dry; may show Xanthodermia</td>
<td>sweating; may show Xanthodermia</td>
<td>dry; urea frost and crystallization on skin pruritis</td>
</tr>
<tr>
<td>Dehydration</td>
<td>+++</td>
<td>not usually present</td>
<td>+++</td>
</tr>
<tr>
<td>Blood Pressure</td>
<td>normal or elevated</td>
<td>normal, low or elevated</td>
<td>elevated</td>
</tr>
<tr>
<td>Temperature</td>
<td>normal</td>
<td>subnormal</td>
<td>subnormal</td>
</tr>
<tr>
<td>Attitude</td>
<td>weak and flacid</td>
<td>anxiety, tremors, convulsions</td>
<td>apathetic, asthenic or increased neuromuscular excitability and rarely convulsions</td>
</tr>
<tr>
<td>Onset of Coma</td>
<td>gradual</td>
<td>sudden</td>
<td>gradual</td>
</tr>
<tr>
<td>Breath</td>
<td>fruity and acetone</td>
<td>normal</td>
<td>ammoniacal. A stirring rod dipped in HCl acid held in front of mouth—breath cloudy white with NH₄ Cl</td>
</tr>
</tbody>
</table>

**Laboratory Findings**

<table>
<thead>
<tr>
<th>Urine—</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific Gravity</td>
<td>high</td>
<td>normal</td>
</tr>
<tr>
<td>Sugar</td>
<td>+++</td>
<td>not in 2nd specimen</td>
</tr>
<tr>
<td>Ketones</td>
<td>+++</td>
<td>not in 2nd specimen</td>
</tr>
<tr>
<td>Casts</td>
<td>depends on arteriosclerosis—in frequent</td>
<td>present in kidney infrequent</td>
</tr>
<tr>
<td>Albumen</td>
<td>+ or —</td>
<td>+ or —</td>
</tr>
<tr>
<td>PSP Test</td>
<td>normal</td>
<td>normal</td>
</tr>
<tr>
<td>Urea Clearance</td>
<td>normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Blood Chemistry—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sugar</td>
<td>high</td>
<td>low</td>
</tr>
<tr>
<td>NPN</td>
<td>occasionally moderately elevated or Normal</td>
<td>normal</td>
</tr>
<tr>
<td>CO₂ Combining Power</td>
<td>decreased</td>
<td>normal</td>
</tr>
</tbody>
</table>
Blood chemistry indicates an azotemia, low sodium, high potassium and sulphate estimations.

The onset of hepatic insufficiency may be sudden as in the hepatorenal syndrome but it is more commonly insidious progressing over many years. Jaundice, ascites, a caput medusae, haemorrhage from rectal or oesophageal varicosities may be evident at time of examination. The fall in cholesterol esters and urea in the blood and the rise of the plasma phosphatase conclude the diagnosis of hepatic insufficiency. Hepatic function tests may also show severe parenchymatous destruction. Coma is often a terminal event in liver failure.
Poisonings—Investigation of the environs for medicine, hypodermics, alcohol or a specific poison is especially important in suspected cases. There are also qualitative tests of patient's urine and blood that may verify the presence of a poison. Complicating a poisoning there may exist head injury or cerebral apoplexy and the clinical picture is quite confused (Table VII).

CARDIO-VASCULAR

All the conditions listed under this heading produce coma largely through cerebral ischaemia. The signs and symptoms of cardiac failure and haemorrhage are too well known to be discussed here. In both, coma occurs near extremis and treatment must be immediate. The panting, cyanotic cardiac and the air-hungry pale bleeder are vivid entities rarely confused with anything else. When embolism or apoplexy intervenes in congestive failure the picture is complicated but the distinct signs of each are present and indicative.

The Stokes-Adams syndrome is a paroxysmal syncope, occasionally associated with muscular twitchings and convulsions, occurring in patients with auriculoventricular heart block. The loss of consciousness is due to cerebral ischaemia and is induced by: (1) ventricular standstill due to vagal inhibition or fatigue of the A-V bundle, or (2) paroxysmal ventricular fibrillation. The attack may last from a few seconds to several minutes. Coma is present after 10 seconds of ventricular standstill or fibrillation; and convulsions appear in 15-20 seconds. The patient is pallid at the start but soon becomes cyanotic. Breathing is stertorous and may be Cheyne-Stokes in type. The neck veins are visibly engorged. Epileptiform convulsions, which eventually follow, are accompanied by incontinence, conjugate ocular deviation, and positive Babinskis. During the seizure the pulse and apex beat are characteristically absent. As the attack subsides, the neurological signs disappear and the pulse rate returns to an even unchanging 40 to 50 beats per minute. An electrocardiogram is confirmatory evidence of the existent heart block.

Carotid sinus syndrome. The patient loses consciousness when undue pressure is exerted on his carotid sinus, as by a tight collar or unusual flexion of the neck, or in periods of emotion and strain. The attack is usually relieved by atropine and adrenaline but not always. The three variations in the syndrome are the vagal, the depressor, and the cerebral types. The vagal group is characterized by ventricular slowing and standstill and relieved by atropine and adrenaline. It is the most common form. The depressor type is marked by a fall in blood pressure, reflex peripheral vasodilatation, unassociated with cardiac slowing. It is relieved by adrenaline but not by atropine. The cerebral type has no bradycardia and no hypotension manifested. It is the rarest and most difficult to distinguish. It is not relieved by atropine or
adrenaline. The patient with any of these three variations recovers when the exciting factor is removed and shows no nervous residua. The diagnosis is based on the brevity of the attack and its rapid defervescence without after effects. An electrocardiogram will show specific effects of sinus pressure in the vagal group only.

**MISCELLANEOUS**

Pneumonia is not an uncommon cause of coma. Coma is present in pneumonic patients most commonly in the extremes of life. The individual is cyanotic, dyspneic and feverish. Signs of consolidation in the chest infrequently are unrevealed by physical examination and roengten diagnosis is required. The picture of a pneumonia severe enough to cause circulatory collapse and coma can be overlooked only with difficulty even by a medical student.

Eclampsia is a convulsive disorder of the latter months of pregnancy or the first days postpartum. The signs of pregnancy or history of recent parturition are always evident. Hypertension, retinal vasospasm, epileptiform convulsions spontaneous or precipitated by the least disturbance, œdema without cardiac failure, and frequent vomiting constitute this toxemia. The urine is small in quantity, grossly smoky, and of a high specific gravity. It contains albumin, red blood cells and casts. The following points are in combination inimitable to eclampsia and make its differentiation from other convulsive comatose conditions easy; occurrence in latter months of pregnancy and early postpartum; signs of pregnancy; repeated convulsions that usually leave no nervous disabilities; œdema without cardiac failure; normal blood urea but high uric acid values; hæmaturia, albuminuria, cylindruria, high specific gravity.

**SUMMARY**

The literature is singularly devoid of papers which discuss comprehensively the diagnosis of the comatose patient. It is for this reason the writer has prepared this review which is a rather practical summary of the signs and diagnosis of coma. Its value lies in establishing a few touchstones characteristic of each condition and offering a foundation to support your own clinical observations.

The author wishes to express his thanks to the Misses Erskine, Messenger and MacKenzie for their invaluable aid in verifying the references, typing the manuscript and reading the galley proofs.

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2. Decreased intake.
3. Impaired intestinal absorption—a condition not infrequently associated in infancy with cretinism, celiac disease, fibrosis of the pancreas and congenital obliteration of the bile duct.
4. Metabolic disorders, as in diabetes mellitus in which the ability of the patient to convert carotene present in foods into vitamin A is greatly reduced.
5. Treatment of conditions due to marked vitamin A deficiency.

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Gallop Rhythm

By RALPH L. PARKER, B.A., '41

INTRODUCTION

THE clinician when employing auscultation in the examination of the heart thinks usually of two heart sounds. However, the presence of a third sound is a more common finding than is generally believed. Thus one should be constantly alert to such additional audible findings, both because of their clinical significance with regard to prognosis, also in their differentiation from extra cardiac sounds.

The presence of a third heart sound or gallop sound has intrigued clinicians almost from the date of the introduction of the stethoscope and so has offered a tremendous field for theorizing regarding its cause, mechanism, and clinical significance.

Despite the extensive writings on the subject, there still remain many unsolved problems.

DEFINITION

Gallop rhythm is the name applied to a peculiar tripling of the heart sounds in a cadence closely simulating the sound of a galloping or cantering horse's hoofs. It has been poetically described as "the cry of the heart for help."

HISTORICAL

Charcellay in 1838 gave an accurate description of an extra heart sound heard in diastole. He attributed it to the contraction of an hypertrophied auricle, and substantiated his theory with post-mortem findings. The term, "gallop," was not used by Charcellay but Brouillard in 1847 designated the condition as "bruit de galop." In 1835, he described reduplication of the first and second heart sounds. Traube finally named this rhythm complex "gallop rhythmus," and although other descriptive terms have been used, "gallop rhythm" has remained firmly entrenched in medical literature. Nevertheless, it was Petain who clarified the maze of confusing terms used by earlier writers. His classical description of typical gallop rhythm and its differentiation from other extra heart sounds remains almost without addition.

In Britain the term gallop rhythm was not employed. In the early literature extra heart sounds were described as "true or spurious reduplications." Spurious reduplications corresponded to gallop rhythm. Later the term "canter rhythm" was introduced, since the rhythm was said to simulate more closely a canter gait than a gallop. However, the previous term, "gallop rhythm," has remained more popular and is used universally.
An extensive review of the literature has been made recently by Holt. Other publications by Duchosal, Mond and Oppenheimer, Bramwell, Wolferth and Margolies, and Thompson and Levine bring the literature up to date.

CLASSIFICATION OF GALLOP RHYTHM AND EXTRA HEART SOUNDS

Many classifications have been proposed for gallop rhythm. The earliest probably was that of Petain, who in clarifying the confusion that existed at the time, classified gallop rhythm as (1) Protodiastolic, (2) Mesodiastolic, (3) Presystolic and (4) Systolic.

Duchosal varied the classification by combining Protodiastolic and Mesodiastolic into Auricular gallop rhythm, while Presystolic gallop rhythm maintained its former position.

Bramwell has attempted recently to clarify the issue by stating, “the so-called protodiastolic gallop is nothing more nor less than accentuation of the physiological third heart sound. If this view is correct it is clearly desirable to dispense with the term ‘protodiastolic gallop’ and reserve the term ‘gallop’ for the presystolic variety. The latter is a distinct clinical entity, and carries a grave significance in prognosis, which is not attached to any other type of triple rhythm.”

Wolferth and Margolies have introduced the term “summation gallop,” in place of mesodiastolic gallop. The reason for this change being the fact that with an increased heart rate presystolic gallop moves farther back into diastole becoming protodiastolic with superimposition of the presystolic sound upon the sound of early diastole.

The classification of Wolferth and Margolies appears to be the simplest and easiest to understand. Therefore their classification of extra heart sounds and gallop rhythm will be used in this paper as a basis for discussion.

1. Reduplicated Heart Sounds:
   (a) Reduplicated first sound,
   (b) Reduplicated second sound.

2. Sounds occurring between the First and Second Sounds:
   (a) Midsystolic clicks,
   (b) Systolic gallop rhythm.

3. Sounds occurring between Second and First Sound (ventricular diastole):
   (a) Opening snap of Mitral Stenosis,
   (b) Physiological third Heart Sound,
   (c) Gallop Rhythm,
      (1) Protodiastolic,
      (2) Presystolic,
      (3) Summation.
REDUPLICATED FIRST SOUND

Many explanations have been offered for this phenomenon.

(1) Asynchronous closure of the mitral and tricuspid valves due to defects in intraventricular conduction.

(2) Defects in auriculo-ventricular conduction.

(3) Asynchronism of closure of the A-V valves is due to delayed contraction of the papillary muscles. (Norris and Landis.)

It has been reported that reduplicated first sounds occur commonly in normal individuals. The recent work of Katz has substantiated this clinical finding. He has shown that in animals there may be asynchronism in the ventricular contraction, particularly as the ejection phase is initiated.

On auscultation, the sound is best heard with the patient seated or erect and at the apex or lower sternum or between the two. The two components of the sound are said to be equal in intensity but occasionally one may be louder than the other. The duration of each component of sound is short. Norris and Landis picturesquely describe the heart rhythm as "k-lub dup; k-lub dup."

REDUPLICATED SECOND SOUND

The mechanism of production of a splitting of the second heart sound is said to be due to two factors:

(1) Asynchronous closure of the aortic and pulmonic valves due to unequal pressure relationships, and/or

(2) Asynchronous closure of separate leaflets of the semi-lunar valves due to an increase in tension of one or more of the leaflets.

Likewise, reduplicated second heart sounds are heard in many normal individuals but in such instances the reduplication can be heard only during a part of the respiratory cycle.

Auscultation of the split second sound is made best at the base of the heart, usually at the second or third interspace. Rarely, it may propagate to the lower end of the sternum. The components of the split second sound are short and sharp with an extremely short interval between them—0.05 seconds.

Norris and Landis describe the rhythm as "lub dub-l; lub dub-l." In some instances the two components may run together and so give a prolonged sound resembling a diastolic murmur at the base of the heart.

MIDSYSTOLIC CLICKS

White has described these peculiar midsystolic twanging noises as "academic curiosities." Huchard attributed these extra sounds to
anomalous chordae tendinae and supported the theory by diagnosing several cases ante-mortem. Some workers have considered this mid-systolic sound of varying intensity as a separate type of gallop rhythm. Wolferth and Margolies set forth the theory that such sounds are extra-cardiac and perhaps due to some fine adhesions of the pericardium to the heart.

The sounds are usually heard nearer the first than the second sound and they vary greatly in intensity and position. Usually they are heard best at the apex but may, in rare instances, be audible over the entire precondium. They have never been proven to be of any clinical significance.

**OPENING SNAP OF MITRAL STENOSIS**

The opening snap of mitral stenosis has been neglected by clinicians regardless of its value as an important adjunct in the diagnosis of mitral stenosis. This can be attributed to the attempts on the part of clinicians to read other sounds into the auscultory findings of mitral valve disease.

This phenomenon of mitral stenosis was commonly described by French authors but received little or no attention in the American literature. Wolferth and Margolies made an excellent study of the French, "Claquement d'Ouverture de la Mitrale," and stated that because its auscultory findings were so characteristic it should be simple to detect. The sound has been described as short, sharp and following the beginning of the second sound by 0.06-0.11 seconds, but the time relationship depends upon the cardiac rate. It is heard best just medial to and above the apex. Rarely it can be heard in the back. In a series of patients with mitral stenosis, Wolferth and Margolies detected the sound in 50 per cent of the cases.

**PHYSIOLOGICAL THIRD HEART SOUND**

Our present knowledge of the physiological third heart sound is due to the extensive work which has been done by Thayer. He reported that 65 per cent of a series of 231 healthy individuals under 40 years of age exhibited this extra sound. He devised certain procedures for accentuating it, e.g., exercise and holding the limbs up with the patient in the supine position. In experiments on dogs, he was able also to produce a sound similar in character to that heard in the human subject. The mechanism of the sound was shown to coincide with the opening of the A-V valves.

Bramwell claims that owing to increased venous return to the heart, the velocity of the blood is increased and is under greater pressure in passing through the A-V orifices. Thus the leaflets of the A-V valves are set into vibration and the third physiological sound is produced. When blood passes without obstruction from the auricle to the
ventricle, no extra sound occurs. The presence of any obstruction, either real or relative, accentuates the third sound. He substantiates his argument with the usual finding of a third sound in mitral stenosis. He states further that the above mechanism is similar to that producing gallop sounds.

The third physiological heart sound occurs in children and young adults. It is associated in no way with cardiac disease or other cardiovascular disturbance. On auscultation, the sound is best heard with the patient in the left, lateral, decubitus position. It is characteristically dull and low pitched and best heard at the apex. In the majority of cases, the sound bears a definite relationship to this preceding second sound, since the mitral valve opens approximately 0.1 second after the aortic valve closes.

**GALLOP RHYTHM**

Gallop rhythm is considered by clinicians as a direct indication of heart failure or myocardial disease.

1. **Mechanism of Production**

A great deal of controversy has arisen over the mechanism of production of gallop sounds. One of the earliest theories advanced was that of asynchronism. The asynchronism was said to involve the complete right and left ventricles, closure of the A-V valves or hemisystole of the ventricles. Following extensive research by workers on the subject, the theory was gradually discarded.

Charcellay's theory that the gallop rhythm sound was due to auricular contraction has remained as a logical explanation of this phenomenon, and is the basis for the present conception. His theory was supported by the fact that the sound was heard best over the auricle and corresponded with auricular systole.

The present conception of its origin is that it may be due to: (1) Auricular contraction, (2) Vibration of the A-V valves or (3) Sudden distension of the ventricular wall with vibration. All experimenters are agreed that auricular contraction is of prime importance in the mechanism but there is much debate on the rôle played by the other factors mentioned. Thus, Bramwell implicates all three factors in the production of the gallop sound. Wolferth and Margolies, on the other hand, regard the vibration of the suddenly distended ventricular musculature as the important accessory factor. They argue that the gallop sound is analogous to the physiological third heart sound and perhaps is produced by a similar mechanism. Therefore they claim the ventricular vibrations are due to some inherent property of the myocardium of young healthy individuals or to the flabbiness of the myocardium when severely damaged.

To summarize, the gallop sound is due to an interplay of possibly
three factors—a fundamental auricular contraction with vibration of the A-V leaflets and/or vibration of the ventricular myocardium.

2. Characteristics of the Gallop Rhythm

In the early literature, gallop rhythm was reported as usually detected in the terminal stages of chronic glomerulonephritis. Nevertheless, it is a common occurrence in coronary occlusion, hypertensive cardiovascular disease, and acute rheumatic carditis. Infectious diseases with myocardial damage may also cause gallop rhythm. Most important of these are pneumonia and diphtheria. Gallop rhythm is seen also in such conditions as severe tachycardia, thyrotoxicosis, anemia, and compensated aortic insufficiency.

Gallop rhythm originating in the right side of the heart occurs with emphysema, pneumoconiosis and asthma.

It is interesting to note that gallop rhythm is rarely, if ever, heard with mitral stenosis.

The time relationship of the gallop sound is dependent upon the cardiac rate. This alone may determine whether or not the gallop rhythm is protodiastolic, presystolic or of the summation variety.

Protodiastolic gallop rhythm is so named because the third heart sound follows the second sound by approximately 0.15 second. Presystolic gallop rhythm is designated as such because it precedes the first heart sound by a very short time interval. It is said to follow the P-wave of the electrocardiogram by 0.08-0.14 second. Summation gallop rhythm occurs during the mesodiastolic period but encroaches upon the protodiastolic and presystolic periods of diastole. Tachycardia is the underlying factor in the production of the summation type of gallop rhythm.

Systolic gallop rhythm has been reported by Thompson and Levine and occurs in mid-systole. It is seen chiefly in individuals not suffering from heart disease. It is more common than is generally supposed.

On auscultation, the gallop sound is dull and thud-like and of variable intensity. It may be so soft that it is completely unrecognized by the clinician. On the other hand, it may be as loud as either heart sounds but rarely louder. A characteristic feature of the sound is its waxing and waning with the phases of respiration. The sound is heard best at the end of expiration, in the left, lateral, decubitus, at or slightly medial to the apex. It may completely disappear at the beginning of inspiration. Usually the sound is confined to a small area. When loud, it may propagate to the lower end of the sternum. Some writers have stated that the gallop sound causes a “shock” which is better palpated than heard. A disconcerting feature of gallop rhythm is its unexplan-
able disappearance. It may be present upon initial auscultation of the heart only to disappear within a short space of time.

Gallop rhythm is heard best in the presence of a moderate tachycardia. However, when the rate exceeds 130 per minute, diastole becomes so shortened that it is extremely difficult to appreciate this extra sound with any degree of certainty. In many instances, exercise brings out the gallop.

3. Clinical Significance

Diastolic gallop rhythm has always been considered a dire prognostic sign.

In a recent review by Thompson and Levine, the average duration of life, after the appearance of gallop rhythm, was eleven months.

If the gallop rhythm clears with the condition causing it, the prognosis is infinitely better. If the condition “resolves” but the rhythm remains, the situation is grave. Thus the disappearance of gallop rhythm in coronary occlusion considerably lessens the gravity of the outcome. Furthermore, in many cases of hypertensive and arteriosclerotic patients, the gallop rhythm disappears completely when the decompensating myocardium is adequately treated. The prognosis is grave in those cases of congestive heart disease with gallop rhythm, especially if there is a previous history of decompensation.

According to Thompson and Levine’s figures, the younger the age at which gallop rhythm is found, the shorter the duration of life. Patients with a blood pressure of 150 mm. Hg., or more, exhibiting gallop rhythm lived longer than those with hypotension.

The occurrence of gallop rhythm in acute rheumatic carditis is uncommon but when present it is of extremely grave prognostic significance. Thus in Thompson and Levine’s series the average duration of life following detection of gallop rhythm was approximately two months.

Systolic gallop rhythm is of no prognostic significance.

4. Differential Diagnosis

Many of the extra heart sounds, and occasionally middiastolic and presystolic murmurs have been misinterpreted as gallop rhythm. Inasmuch as the latter is of great importance, it becomes necessary for one to be capable of differentiating the various sounds. In some cases, this is no easy matter.

The differentiation of presystolic gallop rhythm from reduplicated first heart sound is difficult. In the former, the extra sound precedes the first heart sound. In the latter, it follows the initial component of the first sound with the two components usually equal in intensity and
each of short duration. Both gallop rhythm and reduplicated heart sound are best heard at the apex. Reduplicated first sound is best heard with the patient seated or erect, while gallop rhythm has maximal audibility in the left lateral decubitus. The presence of cardiac enlargement favors a diagnosis of gallop, since reduplicated first sound is commonly found in healthy individuals with no cardiac disease.

Reduplicated second heart sound is less difficult to differentiate. The only possibility of confusing the reduplicated sound with gallop rhythm would be in the protodiastolic and summation types of gallop. The reduplicated second when present is best heard at the base. The sound is of short duration, sharp quality and high pitch, while that of gallop rhythm appears at the base and is a low-pitched, dull, thud-like sound.

Midsystolic clicks occurring between the first and second heart sounds have none of the characteristics of gallop rhythm. To the uninitiated, however, they may be confused with systolic gallop rhythm.

The opening snap of mitral stenosis is differentiated primarily because gallop rhythm rarely, if ever, occurs in mitral stenosis. The opening snap occurs in 50 per cent of the cases of mitral stenosis. It is best heard medial to and above the apical beat and is of a short, snapping character.

The physiological third heart sound cannot be differentiated from gallop rhythm by the usual procedures. Both sounds are apparently produced by similar mechanisms. The auscultatory findings of each are nearly identical in quality, time relationship and pitch. They are both best heard in the same area. The one criterion for diagnosis is the state of the cardiac condition. Therefore in the face of myocardial damage, manifested clinically by cardiac enlargement, gallop rhythm rather than physiological third heart sound may be safely diagnosed. In addition, the third sound is common in normal, healthy children and young adults. It is seldom present in healthy middle-aged patients, so that above the age of 40 the third heart sound is not taken into consideration. However, gallop rhythm can occur at any age, its only prerequisite being alteration of cardiac function due to myocardial disease.

The study of gallop rhythm is indeed fascinating and as yet far from complete. No doubt the future will add further to our knowledge of this interesting auscultatory accompaniment of heart disease.

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Recent Studies of Circulation Time

By J. G. Renegar, '41 and J. W. Babb, '41

The circulation time test is a means of estimating circulatory efficiency and can be used as an aid in the differential diagnosis of certain cardiac conditions. A review of the recent work done on this subject is presented in this article.

For practical clinical purposes, the venous circulation time and in some cases a part of the arterial circulation time are observed. The methods of measurement consist of the injection of a substance into an arm vein at the elbow and observing the time taken for it to be detected by the patient in another part of the body.

The more common tests can be divided into two groups: (1) those appreciated at the base of the tongue (arm-to-tongue test), and (2) those appreciated in the lung (arm-to-lung test). Substances used for the arm-to-tongue test are calcium gluconate, magnesium sulphate, decholin, and saccharin; those for the arm-to-lung test are ether or paraldehyde. Details of the technique are outlined below.

Plotz found the ether (arm-to-lung) test to be of great value in differentiating "asthmatoic heart failure" from bronchial asthma. The latter disease, of an allergic nature, seldom occurs for the first time in a patient over 60 years of age. The main condition likely to be confused with bronchial asthma at such an age is heart failure. Some forms of left ventricular failure produce a paroxysmal dyspnoea indistinguishable, as regards the symptoms or therapeutic response, from bronchial asthma. Occasionally, during an attack of asthmatoic respiration, an accurate diagnosis is impossible and it is here that the circulation time test is of value. A differential diagnosis by means of the venous pressure test would seem simpler but it has two drawbacks. Some cases of left ventricular failure have no increase in venous pressure and during an acute paroxysm the venous pressure of an asthmatic patient is usually sharply elevated, regardless of the cause.

In differentiating cardiac from bronchial asthma, the ether arm-to-lung test has been found to be quite satisfactory. Three-tenths of a cc. of ether and an equal amount of normal saline were injected rapidly into an arm vein and the length of time elapsing until the ether vapor was perceived subjectively was measured. During a paroxysm the circulation velocity of the asthmatics was increased. This makes the circulation time test even more sensitive, since it accentuates the differences in the blood velocities in bronchial and cardiac asthma.

Bernstein and Simpkins reported on the use of magnesium sulphate in measuring the circulation time. Their technique consisted in
placing the patient in the supine position, with the arm on a level with the right auricle. Six cc. of 10 per cent magnesium sulphate were injected as rapidly as possible into an arm vein. The end point was marked by a sudden sensation of heat in the tongue and pharynx, which afterwards passed with the arterial circulation into all parts of the body and disappeared in 10 to 20 seconds.

No untoward results were encountered but in the case of a reaction calcium gluconate was the antidote. The average normal time was found to be 12.9 seconds, with a range of 7.0 to 17.8 seconds.

The authors found no variation beyond normal limits due to weight, height, blood pressure or pulse rate. Applied to various heart cases, it was found that the circulation time increased somewhat parallel with the degree of decompensation. The circulation time was found to be strikingly decreased in hyperthyroidism and was of value in the diagnosis of obscure cases of this malady. Cases of cardiac infarction showed no change. The test may be of value in differentiating hepatic enlargement of cardiac origin from that due to other causes. In a series of some 500 cases, the authors found no nocuous results from the use of magnesium sulphate.

Sigler, Nash, Stein and Epstein attempted to correlate the clinical, electrocardiographic and circulation time findings in estimating the cardiac status during infectious diseases. The question of how frequently the heart is damaged during the course of infectious diseases and how much such damage contributes to the symptomatology has not as yet been definitely answered.

During the past few years an effort has been made to answer this question by means of the electrocardiograph. More recently, studies of the circulation time have been used for the same purpose. The circulation time was determined by the saccharin and ether methods at the height of the disease, or when the symptoms were marked. Electrocardiograms were obtained at the same time; also, the clinical features such as murmurs, cyanosis and dyspnoea were recorded. The normal limits for the circulation time were set at nine to 16 seconds for the saccharin time, and 3.5 to 8.0 seconds for the ether time. The circulation time was more often prolonged among those patients who died and those over 45 years of age than among those who survived. Normal or abnormal circulation time did not go hand in hand with corresponding changes in the electrocardiogram. Marked changes often occurred in one, while the other was quite normal. Furthermore, no relation was noted between abnormalities in the electrocardiogram and clinical signs of dyspnoea, cyanosis, abnormal heart sounds and murmurs (during infectious diseases). However, prolonged circulation time occurred more frequently in the presence of these symptoms.
It appears from the result of the above studies that, although changes in the electrocardiogram frequently occur in infectious disease, they may not signify any cardiac involvement. It may be that such changes are due in many cases to the febrile state itself, and in pneumonia to such conditions as alterations in the environmental media of the heart and slight positional changes of that organ caused by solidification of the lung. The circulation time appears to be a much better criterion of the presence of cardiac involvement in infectious disease than the electrocardiogram.

Manchester advocates the use of paraldehyde in the arm-to-lung circulation time test. His technique consists in the injection of 0.5 cc. of undiluted paraldehyde (U.S.P.) into the antecubital vein. The endpoint is the perception of the odor of paraldehyde both by the patient and the examiner. The patient must be warned to expect a transient paroxysm of coughing. In normal subjects, the time was found to be between nine and 16 seconds. The test may be safely repeated in from five to 10 minutes. The paraldehyde must be of pure quality kept in a well-stoppered bottle and stored in a dark place. Otherwise it will degenerate to acetaldehyde, which is toxic. Manchester experienced no difficulty in the use of this method. His results conform to the findings of other workers.

Baer regards the circulation time as one of the best means of estimating the circulatory status of a patient and of differentiating cardiac, pulmonary and extra thoracic causes of dyspnoea. He uses the calcium gluconate method as described above, except that four cc. instead of five cc. of the solution is employed. By means of the same needle, the ether method is then carried out. He has found the normal range for the ether method to be three to eight seconds. He also found the normal range for the calcium gluconate method (arm-to-tongue) to be eight to 16 seconds and he feels that the calcium gluconate method is the most desirable procedure to date for determining the arm-to-tongue time.

The widest application of these tests is in the investigation of patients with cardiac disease. The difference between the arm-to-lung and the arm-to-tongue time (lung-to-tongue time) is of value in localizing the part of the circulation in which blood flow is retarded. With a normal arm-to-lung time there can be no right heart failure; whereas with a prolonged lung-to-tongue time there must be a retardation somewhere between the pulmonary veins and the tongue, that is, the left heart. Baer presents several case reports which confirm previous findings that circulation time is approximately doubled in the presence of congestive heart failure. He also found this method to be of value as an index of the cardiac response to therapeutic measures.
EXPERIMENTAL RESULTS

The following are the results of circulation time tests performed on a group of patients in Victoria Hospital, London. Two tests were done on each patient, namely, the ether or arm-to-lung test, and the magnesium sulphate or arm-to-tongue test. The normal value for the ether test is three to eight seconds, while that of the magnesium sulphate test is seven to 18 seconds.

**Case 1.** Mr. F., aged 39; complete heart block. The ventricular rate was 40 and the auricular rate 250 per minute.

Ether time—five seconds. Magnesium sulphate time—12 seconds. These observations suggest that, in spite of complete heart block, the circulation time can be essentially normal.

**Case 2.** Mr. S., aged 75; generalized arteriosclerosis with intermittent claudication and attacks of auricular fibrillation.

Ether time—8.5 seconds. Magnesium sulphate time—17 seconds. In this case the arm-to-lung time was somewhat more prolonged than the arm-to-tongue time, suggesting a preponderance of right-sided heart damage. The fact that both tests were near the upper limits of normal indicates a limited amount of cardiac reserve.

**Case 3.** Mr. D., aged 28; rheumatoid arthritis and mitral endocarditis.

Ether time—six seconds. Magnesium sulphate time—14.3 seconds. These values were well within the normal limits and confirmed the electrocardiographic findings that the cardiac damage was minimal.

**Case 4.** Mr. W., aged 70; admitted with pulmonary oedema due to cardiac decompensation. The ether circulation time test shortly after admission was 11 seconds. Subsequently while the patient was receiving digitalis the time was six seconds. However, the magnesium sulphate arm-to-tongue time was 22.3 seconds. These times indicate that his decompensation was primarily left-sided since the right-sided time has come down to normal, while the left-sided time remained prolonged.

**Case 5.** Mr. T., aged 34; asthmatic attacks for 31 years; dyspnoea and palpitation on exertion suggestive of cardiac disability.

Ether time—four seconds. Magnesium sulphate time—12 seconds. Such normal values tend to rule out any cardiac failure.

**Case 6.** Mr. M., aged 54; anginal syndrome.

Ether time—seven seconds. Magnesium sulphate time—27 seconds. The normal arm-to-lung time (right heart) and the prolonged arm-to-tongue time (right heart plus left heart) indicated that the retardation of blood velocity was in the left heart, thus localizing the myocardial pathology to the left side of the heart.
Case 7. Mr. S., aged 87; cardiac failure (arterio-sclerotic basis).
Ether time—six seconds. Magnesium sulphate time—20 seconds. These results are similar to those for the previous case, indicating primarily left-sided decompensation.

Case 8. Mr. M., aged 61; extra-systoles; hepatomegaly of unknown origin.
Ether time—seven seconds. Magnesium sulphate time—16.3 seconds. These circulation times tend to exclude the heart as a possible cause of the enlargement of the liver.

Case 9. Mr. S., aged 68; hypertension; arterio-sclerosis; anaemia (secondary); cardiac hypertrophy.
Ether time—8.5 seconds. Magnesium sulphate time—14 seconds. Anaemia usually increases blood velocity, but in this case the circulation time was slightly prolonged through the right side of the heart. Perhaps the accelerating effect of the anaemia was counteracted by the hypertension.

Case 10. Mr. L., aged 63; cardiac decompensation; grossly enlarged heart, aortic regurgitation.
Ether time—five seconds. Magnesium sulphate time—20.5 seconds. These values show that the heart failure was primarily left-sided.

SUMMARY
1. The circulation time is prolonged in congestive heart failure, and decreases with restoration of compensation.
2. When the left heart, principally, is in failure the arm-to-tongue time is prolonged, while the arm-to-lung time may be within normal limits, or is increased to a lesser degree.
3. The arm-to-lung time is a measurement of the functional state of the right ventricle. The arm-to-tongue minus the arm-to-lung time is equal to lung-to-tongue, which is a measurement of the left ventricular functional capacity.
4. In pulmonary and mediastinal disease the circulation time is not prolonged (although the venous pressure may be elevated) in our experience. Thus it is a fairly specific measurement of the cardiac function.

REFERENCES
THE SPONTANEOUS HYPOGLYCEMIAS
By J. W. Conn
J.A.M.A., 115:1669, 1940

The author stresses the importance of etiology in determining the treatment of spontaneous hypoglycemia. He presents the following simple classification:

I.—ORGANIC
(Recognizable anatomic lesion)
(a) Hyperinsulinism:
1. Pancreatic island cell carcinoma.
2. Pancreatic island cell adenoma.
(b) Hepatic disease:
1. Ascending infectious cholangiitis.
2. Toxic hepatitis.
3. Diffuse carcinomatosis.
4. Fatty degeneration.
5. Glycogenosis (Von Gierke's disease).
(c) Pituitary hypofunction (anterior lobe):
1. Destructive lesions (chromophobe tumours, cysts, etc.).
2. Atrophy and degeneration (Simmonds' disease).
3. Thyroid hypofunction (? secondary to pituitary hypofunction).
(d) Adrenal hypofunction (cortex):
1. Idiopathic cortical atrophy.
2. Destructive infectious granulomas.
3. Destructive neoplasms.
(e) Central nervous system lesions.

II.—FUNCTIONAL
(No recognizable anatomic lesion)
(a) Hyperinsulinism (? autonomic nervous system unbalance).
(b) Renal glycosuria.
(c) Severe continuous muscular work.
(d) Pregnancy and lactation.

From 80-90 per cent of all cases of spontaneous hypoglycemia fall into three groups, namely, functional hyperinsulinism, organic hyperinsulinism, and hepatic disease. With these in mind, a diagnosis may be attempted by study of:

(a) The fasting blood sugar level—This is an aid in separating functional hyperinsulinism from the organic causes of spontaneous hypoglycemia. In the functional condition, the fasting blood sugar is at a relatively normal level.

(b) The dextrose tolerance test—In both organic and functional hyperinsulinism not associated with previous undernutrition or liver disease, the dextrose tolerance response is characteristically of the low type. However, in hepatic disease, the test yields a high plateau type of curve similar to that seen in diabetes mellitus, with the exception that the fasting level is usually abnormally low.

(c) Tests of liver function—The standard Bromsulphalein, hippuric acid, galactose tolerance, urinary urobilinogen, serum proteins and cholecystographic studies are used.

(d) Observation of clinical course—In organic hyperinsulinism and hepatic disease the course is progressive, and attacks become more frequent and severe. Functional hyperinsulinism exhibits no tendency to become more severe.

Hypofunction of anterior pituitary and adrenal cortex and renal glycosurias should also be kept in mind as causes of fasting hypoglycemia.

Indications are given for the different types of surgical treatment and the medical management of the more important causes outlined.

Dr. Conn is an authority in the field and his article makes interesting and educational reading.

—M. Nareff, '42.
THE ETIOLOGY AND PATHOGENESIS OF CORONARY ARTERY OCCLUSION

By A. C. Derby

McGill Journal 10:27, 1940

In about 25 per cent of occluded coronary arteries no autopsy evidence of infarction is present. Ischemia of the myocardium invariably follows occlusion; other factors operate before actual myocardial necrosis occurs. These are: (1) the rapidity with which occlusion develops; (2) arteriosclerosis of the collateral circulation; (3) anemia; (4) lowered blood pressure (shock; low diastolic B.P. of aortic insufficiency); (5) if, after “an attack,” the demands on the myocardium are quickly reduced by rest in bed, sedatives or the control of the rapid ventricular rate, infarction can be prevented; (6) if the heart is hypertrophied and decompensated it is unable to withstand occlusion well because of the relative reduction of the blood supply to the myocardium in an hypertrophied heart and also because the blood supply to a decompensated heart has a low oxygen tension.

There are three conditions commonly associated with coronary artery occlusion. The most important is atherosclerosis of the coronary arteries, with or without thrombosis, which forms over 90 per cent of acute coronary deaths. The exciting factor is a rupture of intimal capillaries which arise directly from the lumen of the coronary arteries. These capillaries arise in response to a thickening of the intima by arteriosclerosis—the blood supply from the vasa vasorum being inadequate under these circumstances.

—Louis Lager, '42.

CRITERIA FOR THE CLASSIFICATION AND DIAGNOSIS OF PERIPHERAL VASCULAR DISEASES

By Salano, Klein, Zurrow, Gootnick and Katz

Arch. of I. M., 25:1035, 1940

A comprehensive diagnosis of peripheral vascular disease should include: A—etiology, B—anatomic status, C—extent of physiologic impairment, D—the functional capacity of the patient.

A—ETIOLOGY:

Acrocyanosis, Arteriosclerosis Obliterans, Arteriovenous Fistula, Autohæmagglutination, Diseases of C.N.S., Cervical Rib Syndrome, Exposure to Cold, Diabetes Mellitus, Embolism and Thrombosis, Endocrine Dysfunction, Ergotism, Erythrocyanosis, Erythromelalgia, Phlebitis, Polycythemia, Raynaud’s Disease and Raynaud’s Phenomenon, Scleroderma, Thromboangiitis Obliterans.

B—ANATOMIC STATUS OF VESSELS AND TISSUES:

1. Vascular Anatomic Status:
   (a) Pallor and rubrocyanosis
   (b) Palpation of pulses
ABSTRACTS

(c) Temperature of extremities by palpation
(d) Oscillometer
(e) Roentgenographic examination.

2. Anatomic Status of Tissues:
(a) No changes
(b) Trophic changes without lesions
(c) Infections
(d) Gangrene
(e) Loss of members.

C—Physiologic Changes:
1. Intermittent claudication
2. Spasm
3. Vascular reserve.

D—Functional Capacity of Patient:
Class 1. Patients with organic disease without symptoms.
Class 2. Patients who have organic disease
   (a) with minimal symptoms
   (b) with moderate symptoms.
Class 3. Patients who have organic disease and are bedridden because of severe pain, gangrene or infection.
Class 4. Patients who have symptoms without organic disease; "functional" group.

THE EFFECT OF DECREASED BAROMETRIC PRESSURE ON THE ELECTROCARDIOGRAM
By C. O. Benson
J. of Av. Medicine, 11:75, 1940

The authors show that no changes occur in the electrocardiogram of healthy subjects at 30,000 feet with adequate oxygen. Heart rate gradually increases at 8,000 feet and above when breathing air. In healthy subjects up to 20,000 feet without oxygen, changes in the T wave and decreased amplitude of QRS complex occur. They infer that with adequate oxygen supply there need be no harmful results even in pathologic hearts. Thus it is concluded that patients with cardiac disease can be safely transported by air.

MYOCARDIAL INFARCTION IN A YOUNG AVIATOR
By A. Graybiel and R. A. McFarland
J. of Av. Medicine, 11:75, 1940

This is a review of the case history of a young pilot with healed myocardial infarction. He had a history with nothing significant with regard to heart but had been flying constantly in China up to 18,000 feet. He had several narrow escapes and one exhausting forced landing. The only evidence of myocardial infarction rested in the electrocardiographic findings. The pilot was recommended to be given a ground position and this was supported by aviation authorities. The article is of interest as a precedent in reporting that the objective findings of a laboratory test have been accepted as showing unfitness for flying.

PREMONITORY SYMPTOMS OF ACUTE CORONARY OCCLUSION
By Master, Dack and Jaffe
Ann. of Int. Med., 14:1155, 1941

Premontory symptoms could be elicited in 44.2 per cent of 260 patients with acute coronary occlusion. These symptoms were chiefly substernal or precordial pain or discomfort. Fatigue, weakness, gastric distress, dyspnea, palpitation, nervousness and dizziness were often present. The premonitory symptoms were usually within 24 hours of the acute attack and often came as an exacerbation of angina pectoris. The pathologic basis for these symptoms is assumed to be the gradual occlusion of the lumen of a coronary artery to give physiological coronary insufficiency and myocardial ischemia. Immediate bed rest on appearance of the warning symptoms should reduce the mortality rate.

HYSTERIA—SOME COMMON MISCONCEPTIONS
By David Lester
Ann. of Int. Medicine, 14:1248, 1941

This excellent article tries to remove the misconception concerning "hysteria," viz.: 1. that hysteria is more or less identical with malingering.
2. that no symptom is "hysterical" unless it can be mimicked by volition.
3. that the hysterical patient could be well if he would only try.
4. that hysterical patients never harm themselves in any way.
5. that patients with hysteria have a specific, predisposing, constitutional defect.
MEDICAL HISTORY AND THE MEDICAL STUDENT

“He who knows only medicine, knows not even medicine.”

This statement is as true today as it ever was. To study medical science without knowing something of its background, its mistakes and its accomplishments, is like having repeated “dry clinics”—the facts are there but the human interest is not. There are many who study medicine as a pure science just as a mathematician studies calculus. They forget that doctors are not dealing with figures, signs and symbols. We are dealing with human beings, all of whom possess those intangible qualities which go to make up personality. Thus we must give medical practice life, not cold, bare facts.

Our ideas are rarely ever original with us. They have been thought many times before and we only build upon the ground work created by preceding generations. Medical history helps us to understand these builders and their works. History gives us perspective so that we do not learn irrelevant and unimportant detail but, instead, select the fundamental principles and essential points. Only one who is informed with contemporary knowledge can get the most out of medical history. Hence, the two can be interwoven so that we get that balance for which we all strive today.

By our acquaintance with the careers of others we are in a position to judge our own fitness to practice medicine and are shown how we can improve. Hippocrates, Celsus and many others of antiquity studied the lives of their predecessors. In modern times such men as Oliver Wendell Holmes, Osler and Cushing stand out in our minds as untiring readers of medical history. Following the custom of these great men, it is to our advantage as doctors and undergraduates to learn as much as we can of the glorious past of medicine.


—L. RUTTLE.
CLINICAL HEART DISEASE

By S. A. Levine, M.D.

(2nd edit., revised and reset; 495 pp., 109 illustrations. Published by W. B. Saunders Co., 1940)

In his preface the author says:

"The purpose of this book is to present in a simple form the important aspects of the diagnosis, prognosis and treatment of heart disease. No attempt has been made to cover in detail the entire cardiovascular field. Special topics that concern the practitioner which merit emphasis are discussed separately. It is much more important that the physician be able to recognize thyrocardiacs, who are masked as heart patients and suffer invalidism so readily preventable, than to be able to make an early diagnosis of subacute bacterial endocarditis."

An excellent feature of this easily understood book is the personal charm of the author sprinkled so generously throughout its pages. Much emphasis is placed on the practitioner's reliance on carefully and quickly done, simple and inexpensive clinical methods to disentangle the complicated differential diagnosis of various types of heart disease. The reader will also appreciate the presentation of appropriate case histories solved because of careful observation of detail.

—L. Lager, '42.

TREATMENT OF WAR WOUNDS AND FRACTURES

By J. Trueta, M.D.

Foreword: Ernest W. Hey-Groves, M.S., F.R.C.S.

(145 pages, 48 illustrations; Hamish Hamilton Medical Books, London, W.C.1; 1939)

This small book begins with an historical sketch of war wound treatment since ancient times; special mention is made of the closed method advocated first by Winnett-Orr et al. This historical introduction ends with a note on the Spanish War. It was in this war that most of the cases cited in the book were treated.

The subject matter begins with a discussion of immobilization of
all war wounds, chiefly of the extremities, traumata, or traumata associated with fracture. Trueta uses good reasoning which seems physiologically sound. He points out that some selectivity of cases is necessary; also the advantages of transport in wounds immobilized in plaster. This was proven to some extent at Dunkirk, in the present conflict.

The eleven chapters, following this introduction, are devoted to specific regional treatment, such as shoulder, arm, hip, etc.

One of the most remarkable disclosures is that, even though the wound itself, beneath the cast, may be filled with virulent bacteria, including B. Welchii, gas gangrene is very rarely encountered.

Much stress is laid throughout on free drainage of the deeper planes of the wound, with gauze strips or hard rubber tubes. Where the latter are used, they are included in the cast.

—J. S. Winder, '42.

APPLIED PHYSIOLOGY
By SAMSON WRIGHT, M.D., F.R.C.P.
(7th edit., 740 pp.; $7.10; Oxford University Press, London, New York, Toronto; 1940)

The seventh edition of this most valuable bridge between theoretical and applied physiology will be received with wide acclaim by both medical student and practitioner alike. The revision has been drastic with many sections completely re-written or newly added, but the character of the book, that of a physiological guide in clinical studies, has been strictly adhered to.

The new material is of the highest quality and the writer has gained much by his association in an editorial capacity with Section A (111) of British Chemical and Physiological Abstracts which deals with experimental medicine in its widest sense.

The addition of 118 new figures is an important feature of this edition, and, as the author says, are worthy of considerable attention.

As a text-book the reviewer considers this a "must" for both the graduate and undergraduate.

—M. P. Wearing, '42.