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Staff Meeting Report

Errors and Pitfalls in Physical Diagnosis*

An Apology for the Art of Medicine

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Most of the material for this discussion has been collected from the routine checking of physical examinations performed by senior medical clerks in the outpatient department.

The purpose of this discussion is to draw attention to certain common errors and difficulties in physical diagnosis and also to the addition of some new and recently re-investigated signs which are of value in clinical medicine.

The ability to elicit physical signs depends upon skilled observation. This is no science but an art. According to the encyclopedias art is "skill and ability acquired through patient practice and directed towards a definite end whether this be aesthetical, ethical, or useful." A rectal examination can hardly be described as aesthetical or ethical, but it is definitely useful. The art of medicine has been decried by many physicians who wish to place medicine in the category of a pure science. They feel that the "art of medicine" is a term used by older or less academic members of the profession to mean bedside manner while it is also an excuse for them not keeping abreast of the modern developments in their field. Some feel it is nothing more than a big income, pin-stripe suit, carnation, and reassuring hand-patting. Others, invariably academicians, feel that it is the straw of empiricism to which certain physicians clutch to avoid drowning in a sea of investigative and laboratory procedure.

You might think that the field of discovery in physical diagnosis was exhausted. This is not so; each year brings new signs or very old ones revived after careful investigation and evaluation by modern physiological methods. Examples include the effect of respiration on splitting of the pulmonary second sound (P_2), the paradoxical splitting of P_2, the systolic ejection clicks, the opening snap of the tri-

* This is a report given at the Staff Meeting of the University of Minnesota Hospitals on March 1, 1957.

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cuspid valve, and the careful clinical analysis of the jugular venous pulse. Since most of the errors and difficulties lie in the field of cardiology and because most of the newer additions to physical diagnosis are to be found there, the majority of my remarks will be directed to this system.

One of the commonest errors is the diagnosis of congestive heart failure when this is not present. This stems from a fundamental lack of knowledge of hemodynamics and from an inability to estimate clinically the height of the venous pressure. On one occasion not a single clerk from a class of 30 knew or could remember Starling's law of the heart nor its application to clinical medicine. It would appear appropriate at this point to describe the technique of venous pressure measurement. The uppermost level of venous pulsation in the internal jugular vein is determined and the vertical distance above the angle of Louis noted. This site is a convenient reference point as it is usually 5 centimeters from the mid right atrium in any position of the patient. For ease of examination the patient is inclined at an angle of 30 degrees from the horizontal, but if the pressure should be very high, the patient may have to be placed upright in order to find the upper limit of pulsation. All rises in venous pressure are not necessarily due to congestive heart failure and Table 1 lists the conditions

TABLE 1

CAUSES OF AN APPARENT RISE IN JUGULAR VENOUS PRESSURE

1. Hyperkinetic states
2. Increased blood volume
3. Slow heart rate
4. Increased intrathoracic pressure
5. Increased intra-abdominal pressure
6. Increased intra-pericardial pressure
7. Obstruction of the superior vena cava
8. Tricuspid stenosis
9. Space-filling lesions of the right side of the heart
10. Giant A waves
11. Physical exertion
12. Congestive heart failure

associated with a rise. On the other hand a rise in venous pressure is a *sine qua non* for congestive failure. The upper limit of normal pulsation is 3 centimeters above the angle of Louis.

Attention is rarely paid to the nature of the venous pulse in the internal jugular vein. Such an examination can be highly rewarding. The striking visible event in the normal central venous pulse as seen in the neck is the collapse of the great veins coincident with the central arterial pulse wave. This collapse is known as the X descent. In tricuspid insufficiency this collapse is replaced by a

large V wave producing systolic expansion. In tricuspid stenosis, severe pulmonary hypertension, or pulmonary stenosis a giant A wave of presystolic pulse is frequently seen. It resembles the arterial Corrigan so greatly, that it is referred to as the venous Corrigan pulse. In nodal tachycardia and in complete heart block similar waves known as cannon waves may be due to the contraction of the auricle against a closed tricuspid valve. For some time it has been taught that abdominal or hepatic compression will raise the jugular venous pressure in patients with borderline congestive failure, the so-called hepato-jugular reflux. This is incorrect as a rise may be seen in normal individuals and appears due only to upward displacement of the diaphragm, consequent reduction in thoracic size, and reduced venous return to the atrium.

The examination of the pulse is sadly neglected, and very few students ever comment on the quality except when it is very obviously collapsing. Moderate degrees of collapse from high output states or mild aortic insufficiency, the plateau type pulse of aortic stenosis, the pulsus bisferiens of combined aortic valve disease, pulsus paradoxus, and even the small pulse of mitral stenosis are rarely commented on. When pulsus paradoxus is found, many students and clinicians alike hold tenaciously to the belief that it is pathognomonic of pericaradial disease. This is not true for it is seen on forced inspiration in the normal person, in respiratory obstruction, and at times in gross cardiac dilatation.

An interesting and commonly misdiagnosed anomaly is the buckled carotid. This is always mistaken for an aneurysm by the tyro. It is a pulsatile swelling of the common carotid artery usually on the right. It results from elevation of the aortic arch from dynamic unfolding when there is increased pressure or loss of elasticity in the aortic wall. This rise means that the original length of carotid must adapt itself to a shortened distance from the arch to the base of the skull by buckling. It is commonest in obese, kyphotic women with systolic hypertension. In general it would appear to be a benign phenomenon. In young people however it may result from coarctation of the aorta. Many such patients are referred annually from practitioners with the diagnosis of carotid aneurysm.

Failure to detect early clubbing is a common pitfall. A clinical diagnosis of carcinoma of the lung or sub-acute bacterial endocarditis may be supported by the detection of clubbing of recent onset. The earliest signs are obliteration of the normal groove between the base

of the nail and the skin proximal to it and easy subluxation of the root of the nail through the skin over the nail. It should be noted that about 5 per cent of all clubbing is familial and that this type is largely confined to males in whom it makes its appearance at puberty. While on the subject of fingers, it is worth pointing out that each year brings a number of patients referred to the clinic for rheumatoid arthritis when the real diagnosis is painful Heberden's nodes, a manifestation of degenerative arthritis. These nodes, which involve the terminal interphalangeal joints and occasionally the proximal joints as well, are commonest in females and are frequently familial.

Lid retraction and real or apparent exophthalmos are found in normal individuals sufficiently frequently to be a diagnostic pitfall. Lid retraction is found in about 10 per cent of the population. This type always involves the upper lid and is most frequently seen in young females where it is poorly sustained and often voluntary. Severe refractive errors, especially myopia, may also cause lid retraction. Mild exophthalmos is seen in about 7 per cent of the population and is at times distinctly racial. It is a noticeable feature of the Maori people of New Zealand. Severe respiratory distress from asthma, emphysema, or acute pulmonary edema as well as actue anxiety may cause temporary exophthalmos.

Students rarely appear to understand the difference or, for that matter, recognize a difference between peripheral and central cyanosis. Central cyanosis implies arterial oxygen desaturation throughout the entire arterial tree, resulting from a veno-arterial shunt or inadequate oxygenation in the lungs. The cyanotic hue will be seen everywhere, even in the warm mucosae although it will be maximal in the extremities. It can usually be detected when the arterial oxygen saturation falls below 85 per cent. Peripheral cyanosis refers to cyanosis of the extremities or exposed portions without central arterial desaturation. This results either from a low cardiac output or from peripheral vasoconstriction, leading to stagnation of blood in the capillaries and greater oxygen extraction by the tissues. Obviously it will not be present in the warmer areas unless the cardiac output is exceptionally low.

Precordial rib retraction is still misleading students. It is not uncommonly seen over the right ventricle in normal individuals with a thin thoracic wall. It is exaggerated, however, in the same region when there is hypertrophy of the left ventricle alone. The combina-

tion of a forcible apex and a left parasternal systolic retraction may lead to a strong rocking movement in a counter-clockwise direction. When there is gross right ventricular hypertrophy a retraction may be seen in the anterior axillary line which if combined with a parasternal systolic lift gives rise to a rocking movement in a clockwise direction, sometimes referred to as a lateral thoracic jerk. Broadbent's sign is a systolic retraction seen posteriorly over the last two ribs on the left side and suggests adherent pericardium in this region.

Abnormal heart sounds can often be palpated. With the exception of the disastolic shock over the pulmonic area resulting from a greatly accentuated P_2 they are usually completely missed. This must surely rise from inadequate palpation, which I believe is due to too much attention to percussion and too little to the much more informative palpation. The opening snap of mitral stenosis can be felt as a sharp early diastolic impact, the abnormal fourth heart sound as a double apical impact, and the third heart sound as a thudding sensation in diastole at the apex. Students invariably palpate with their finger tips rather than by the correct and more accurate method of using the palm of the hand.

Too much reliance is placed on percussion of the heart borders by students and teachers. Cardiac enlargement is often found by this method when it is not present. Furthermore most students use percussion to the exclusion of good inspection and palpation. The greatest danger of percussion is not its inaccuracy but in its detraction of examiners from using their eyes and touch. Percussion is at its best when the point of maximum impulse (P.M.I.) can be seen or felt and at its worst when the P.M.I. cannot be detected by these means. Percussion gives no idea about the quality of the apex beat and thus no clue to the nature of the underlying ventricular state. Although percussion is a regular routine with them, not one clerk in a whole year recorded any information concerning the quality of the P.M.I. from palpation.

Heart sounds and murmurs still pose the greatest problems in recognition and interpretation for the physician. Students and physicians alike rarely pay much attention to the individual heart sounds themselves unless they listen with a preconceived idea of what they expect to hear. This has been responsible for the failure to detect in the past a number of abnormal sounds which have only recently been emphasized. If, after what he considers a careful cardiac examination, a

student is asked to comment on the heart sounds, he is usually at a loss, yet such an examination may be more rewarding than a description of various murmurs.

The split mitral first sound, which is best heard just to the left of the sternum in the fourth space, is usually normal. It results from the slight delay in closure of the tricuspid valve over the mitral valve. It is often mistaken for the presystolic murmur of mitral stenosis. Such a mistake should not occur but continues to do so. With a split there is no continuous crescendo of sound ending in an accentuated first sound, nor is the helpful opening snap ever present. The split may also be mistaken for a fourth heart sound which gives rise to the so-called presystolic or atrial gallop. The quality of the fourth sound is entirely different from that of the mitral element of the first sound. The former is a thudding sound best heard with the bell, while the latter is shorter, sharper, and of course nearer to the tricuspid element of the first sound.

The opening snap of mitral stenosis still fails to be understood or even detected despite its value either in diagnosis or in determining the degree of associated mitral insufficiency. It is a quite loud, short, high pitched sound best heard in expiration along the left sternal border in the third or fourth intercostal space. It is due to the high atrial pressure suddenly forcing open the stenosed valve when the ventricular pressure falls sufficiently in early diastole. The more severe the stenosis is the earlier the snap as the atrial pressure rise will occur sooner and exceed the ventricular pressure earlier. Its presence is pathogomonic of mitral stenosis and generally rules out any serious degree of mitral insufficiency. It is almost always mistaken for a split P_2. This error would not be made if students realized that a normally intense P_2 cannot be heard in the fourth space whereas marked splitting with an accentuated P_2 is rarely heard. Consequently an apparent split of the second sound along the left sternal border or at the apex when P_2 is otherwise normal is strongly in favor of an opening snap. Occasionally an opening snap is confused with a third heart sound which is very commonly heard in mitral insufficiency. The third sound is dominant at the apex and is low pitched and thus heard best with the bell. The opening snap may, of course, arise from the tricuspid valve in the very much rarer tricuspid stenosis. Then it is heard in the same site but like all tricuspid sounds it is much louder in inspiration.

Splitting of the P_2 can be difficult to understand. The split itself, like the split of the first heart sound, is due to asynchronous closure of the participating valves. Since the aortic valve closes about 0.03 second before the pulmonic, it is obvious that the first element of the split is aortic. The split increases on inspiration and decreases on expiration. The widening of the split appears to be due to increased filling of the right ventricle on inspiration leading to slight prolongation of ventricular systole and thus to later closure of the pulmonary valve. The split is increased in any condition leading to increased filling of the right ventricle or in right bundle branch block (R.B.B.B.). In atrial septal defect the split is fixed since right ventricular filling is maximal with each cycle, but in R.B.B..B. the split still varies some with respiration. In left bundle branch block (L.B.B.B.) the split may behave paradoxically, increasing on expiration and decreasing on inspiration. This results from the aortic element of the second sound occurring after the pulmonic instead of before because of the delayed activation of the left ventricle. Now on expiration the pulmonic sound moves as usual in towards the first heart sound but in doing so widens the split. This phenomenon is also seen in aortic stenosis and other conditions with left ventricular hypertrophy.

Systolic ejection clicks are heard in both aortic and pulmonic areas. They are short sharp sounds occurring about 0.06 to 0.08 second after the sound of the mitral closure. They are apparently vascular in origin and coincident with the opening of the semilunar valves. They are sometimes heard when ejection is hyperdynamic as in thyrotoxicosis, anemia, and emotional states. The aortic ejection sound is usually heard where there is dilatation of the ascending aorta from any cause such as aneurysm or post stenotic dilatation. It is best heard in the third left intercostal space at the sternal edge or at the apex and is most marked in expiration. The pulmonary ejection sound is heard where there is pulmonary hypertension, mild pulmonary stenosis, or dilatation of the pulmonary artery from any cause. It, too, is located at the third interspace but is not audible at the apex. In the past these two sounds have been ignored and written off as split first sounds.

The mid-systolic sounds are three in number. First, there is the dull thud of the systolic gallop which, although of unknown significance, is heard in normal individuals. Second, there is the clicking noise described by Gallavardin possibly associated with pleuro-pericardial adhesions. Finally there is the click of the small left-sided pneumo-

thorax which varies markedly with posture and respiration. These sounds are all benign and should not be confused witth the more significant diastolic extra sounds.

A discussion of the clinical difficulties encountered in the interpretation of murmurs could occupy this whole paper so I must confine my attention to a few of the problems only.

The systolic murmur of aortic stenosis which is at times heard best at the apex may be confused with that of mitral regurgitation. Regurgitant murmurs of the atrio-ventricular valves must obviously be pansystolic, as the leak begins at the onset of isometric contraction. On the other hand ejection murmurs of the semilunar valves begin later in systole with the opening of the semilunar valve itself. This, of course, is the site of the ejection sound which at times may be heard to initiate the murmur. Besides this later start, the ejection murmur rises to a peak in mid-systole unlike the regurgitant murmur which continues unabated throughout the whole of systole. The ejection click which may be loud at the apex in aortic stenosis may be mistaken for the first sound proper and the murmur deemed pansystolic. This mistake will not occur if the student is aware of and familiar with this sound.

The systolic murmur of mitral insufficiency may be confused with that of tricuspid insufficiency. This may lead to a mistaken decision regarding the suitability of the patient for mitral valvotomy. She may be denied surgery when she has nothing more than pure mitral stenosis and functional tricuspid insufficiency. The murmur of tricuspid insufficiency is usually maximal along the left sternal border and is characteristically accentuated by inspiration. The presence of a good opening snap in addition would be good evidence in favor of the absence of significant mitral insufficiency.

The late systolic murmur is undeniably the most confusing murmur in the book. It is audible in the latter part of systole only and appears to have a crescendo quality up to the second sound. When it is first heard it is always mistaken for the presystolic murmur of mitral stenosis. The murmur is actually pansystolic, but the first part is of too low a frequency and intensity to be heard by the human ear. It is either completely innocent or indicates trivial mitral insufficiency. It should not be confused with the pansystolic murmur with a late accentuation which is strongly suggestive of mitral insufficiency. The only other late systolic murmur of importance is that heard over the base or

posteriorly in coarctation and attributed to turbulence at the site of the stricture.

The apical diastolic murmur which is taught as being diagnostic of mitral stenosis is far from being so. Table 2 lists those conditions in which it may be found. Functional apical diastolic murmurs are

TABLE 2

APICAL DIASTOLIC MURMURS

Organic

1. Mitral stenosis
2. Mitral insufficiency
3. Left Atrial Tumour
4. Acute rheumatic fever (Carey Coombs murmur)

Functional

1. Austin Flint
2. Atrial septal defect (probable torrential tricuspid flow)
3. Ventricular septal defect
4. Patent ductus arteriosus
5. Thyrotoxicosis
6. Anemia
7. Complete heart block

(3–7: Probable torrential mitral flow)

8. Coarctation
9. Congenital aortic stenosis

(8–9: Possibly minor fibro-elastotic changes)

commonly found in congenital heart disease especially where there is a left-to-right shunt. This type of murmur is softer than its cousin in mitral stenosis and is probably due to torrential blood flow through the atrio-ventricular valves. In atrial septal defect the increase in flow is through the tricuspid value, and the murmur, as expected, is loudest in inspiration. Torrential mitral flow accounts for the murmur in anemia, complete heart block, ventricular septal defect, and patent ductus arteriosus. The diastolic murmur in acute rheumatic fever, which was described by Carey Coombs and is now known by his name, is a short low pitched diminuendo sound. This does not represent mitral stenosis but is attributed to turbulence set up by inflammatory thickening of the mitral cusps. It subsides rapidly in the convalescent period.

Many people still believe that the peripheral signs of aortic insufficiency are necessary for its diagnosis and that an isolated early diastolic murmur along the left sternal border means pulmonary insufficiency. This is, of course, incorrect. The isolated early diastolic murmur along the left sternal border is most often due to pure aortic insufficiency. In general there must be a good pathological and clinical reason for pulmonary insufficiency, such as marked pulmonary hyper-

tension with mitral stenosis or with congenital defects. The pulmonary murmur does not radiate as does that of aortic insufficiency and is nearly always associated with visible pulsation of the pulmonary artery, an accentuated P_2, and a systolic ejection click.

Not all continuous murmurs are due to patent ductus arteriosus, and Table 3 indicates that the number of causes are far from few. Diffi-

TABLE 3

CONTINUOUS MURMURS IN THE CHEST

1. Patent ductus arteriosus
2. Aorto-pulmonic window
3. Venous hum
4. Lactating breast
5. Ruptured sinus of Valsalva aneurysm
6. Persistent truncus arteriosus
7. Pulmonary atresia
8. Coronary arterio-venous fistula
9. Anomalous drainage of a coronary artery into the atrium
10. Aortic arch syndrome
11. Pulmonary arterio-venous fistula
12. Intercostal arterio-venous fistula

culties are occasionally met in distinguishing a venous hum from the typical Gibson murmur of patent ductus. The hum has been known for a long time, having been described by Potain in 1867, but is still after all these years responsible for mistakes in diagnosis. It is often first noted on auscultating the aortic or pulmonary areas but is best heard over the jugular veins themselves. It is loudest with the patient in the sitting position and usually disappears when he lies flat with the jugular vein collapsed or with execution or a Valsalva maneuver. It is quite common in young people.

To end in a different field, I would like to draw attention to a neurological condition which may cause trouble for the internist. This is the so-called Adie syndrome or the tonic pupil with absent tendon reflexes. This apparently benign condition of unknown etiology is seen almost exclusively in young females. It is characterized by the appearance of a dilated pupil usually only on one side which at first glance reacts neither to light nor convergence. In addition the knee and ankle jerks are absent so that the condition may be mistaken for tabes dorsalis. If however, the patient is asked to converge steadily on some object over a prolonged period of time the pupils will constrict slowly. A similar reaction may be found to light. Recently we saw a young female who had a constrictive type of pericarditis and a dilated fixed pupil on the left. The consensus was that the two conditions were related, that the pericarditis was infiltrative in nature

with cervical sympathetic irritation on the same basis. Careful examination later revealed that the pupil behaved as an Adie pupil, while subsequent thoractomy revealed the benign nature of the constricting lesion of the pericardium.

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Staff Meeting Report

Hemangiomata and Some Associated Congenital Vascular Syndromes*

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During the past century numerous syndromes have been described associating varied types of cutaneous hamangiomata with lesions of the central nervous system, the skeleton, the eye, and other organs. These syndromes are characterized by inconsistencies in their description and more often by the plethora of eponyms given them. Too often authors have described partial or incomplete forms of one or another of these complexities, thus helping confuse the reader.

These congenital defects have held wide interest and present a wide range of clinical findings. We would like to review three entities for purposes of general information and hope thereby to define more clearly some particularly interesting details of them.

The three congenital vascular syndromes are known, respectively, as the Sturge-Weber, Maffucci, and Klippel-Trenaunay syndromes. Since we shall frequently mention the hemangiomatous aspects of these three diseases complexes, however, we should briefly recall some features of hemangiomata or vascular nevi. It is well to avoid the use of the term nevus in speaking of hemangiomata, since many restrict that title to those pigmented lesions of the skin in which nevus cells are demonstrated histologically. We should further refrain from the use of the term nevoid, as this term is without concrete meaning.

The present general classification of hemangiomata is confusing. It includes numerous forms of telangiectasia and acquired vascular defects. This discussion will be limited to the congenital vascular defects only.

We recognize four basic clinical types of hemangiomata. These are the port-wine mark or nevus flammeus, the strawberry type, the cavernous type, and the plexiform or racemose type. The first three types are composed of capillaries and the latter of venules,

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veins, arterioles, or all three. It must be borne in mind that many instances represent mixtures of two or more of these four types.

Type 1. This is the port-wine mark or stain, commonly referred to as the nevus flammeus. Several types of lesions bear this name, but differ from one another in incidence, site, and prognosis. An insignifican type is the lesion which appears on the face in some infants and which soon regresses and disappears in early childhood. Another common form appears in the nuchal region in many newborn infants and often persists into adult life. These lesions tend to be quite small and irregular in configuration and vary in color from pink to light red. These forms of nevus flammeus have no relationship to the syndromes to be presented.

Another less common form of the port-wine mark or nevus flammeus is usually more intense in color and often appears in the distribution of a cutaneous nerve. These frequently have areas of extensive engorgement and thus are turgescent and erecticle. In some instances, despite a suggested distribution in the pattern of a nerve trunk, the hemangioma may not be uniform but may contain areas of apparently normal skin, mixed with areas of the hemangioma in irregular fashion.

Both forms of port-wine mark represent collections of dilated capillaries appearing in increased number per unit of skin area. The capillaries are at the depth of the mid-cutis. The lesions following a cutaneous nerve distribution do not undergo spontaneous involution, and treatment is undesirable because of the resulting scarring.

Type 2. The strawberry mark usually appears shortly after birth. They are composed of clusters and tufts of capillaries and appear as well circumscribed, compressible, elevated lesions, with predilection for the head, neck, and upper torso. They appear more often in female infants. After their appearance they tend to enlarge and usually reach their maximum size in 5 to 10 weeks, at which time spontaneous involution often begins. In the absence of complication or treatment and upon involution no scar will mark its site. When composed of very superficial capillaries, the lesion is elevated and an intense red in color. At times the capillary structure is found deeper in the cutis and contains more blood, because of intimacy with larger vessels. In these forms, the skin appears more nodular, turgescent, and may be likened to erectile tissue. These more deeply placed lesions tend also to be larger, and may cover sizeable areas or include the skin of an entire extremity.

Type 3. The cavernous hemangioma is likewise of capillary structure but is deep in the skin and heavily engorged with blood. These forms are often absent at birth and appear in early infancy. In the pure form there is a bluish tumefaction deep in the skin, with the overlying epidermis appearing stretched and translucent. A mixed superficial and cavernous form is more frequent, and the more superficial portion undergoes spontaneous involution. However, the cavernous portion does not always undergo expected spontaneous involution but will respond well to minimal roentgen therapy or the injection of sclerosing solutions. On involution after treatment a flaccid atrophic saclike scar remains.

Type 4. The fourth type is the plexiform or racemose hemangioma. These are best likened to the plexiform neuroma and are rare. The structure includes venules, veins, and at time arterioles. These have been described under various headings, including the term congenital varicose veins. They are not visible in infancy, but appear in childhood or young adult life. They may or may not have associated overlying cutaneous hemangiomas of varied types.

Numerous arterio-venous communications can appear in the mixed arteriolevenous forms and have received considerable attention in the literature of recent years. It is now vogue, when referring to the forms composed of small veins and venules, to use the term phlebectasia. When arterioles participate in the structure of the hemangiomas, the resulting structures are properly referred to as phlebarteriectases. Some authors group both types and speak of them as vascular hamartomas.

A report in 1952 reviewed the incidence of various hemangiomas in a group of newborn infants. Strawberry mark, or simple angioma, appeared in 10 of a series of 879 infants, nevus flammeus or port-wine stain of the neck in 42 per cent of white infants and in 31 per cent of colored infants. A nevus flammeus, or port-wine mark of the face or limb, appeared in one patient in this series. The plexiform type was not observed.

The origin of hemangiomata was long ascribed to factors such as intra-uterine pressure, "accidents", etc. Embryological studies have shed new light on the formation of blood vessels and from these studies we have more plausible explanations for their existence.

Vascular malformations presumably arise from mesodermal dysplasia. Other dysplasias may occur coincidentally, thus indicating that some embryonic force or forces can simultaneously alter one or more tis-

sues. The normal development of vessels has been shown to occur in three steps: 1. early development of a fine capillary network in the angioblastic mesoderm, 2. development of preferential channels within this capillary structure, and 3. the final investment of the vessels with their muscular and elastic endowment. During the second stage of formation, the blood flow may be alternating in any given vessel, and at this phase it is impossible to delineate primitive vein from artery. The formation of hemangiomata appears to result from localized arrests of development at or between stages. Local arrest may simultaneously occur in formation of other structures or organs, thus giving rise to multiple defects in metameric distribution.

Sturge-Weber Syndrome

This is the best known of the three syndromes. Credit for the earliest description in 1860 has been given Schirmer. Sturge's description appeared in 1879 under the title, "A Case of Partial Epilepsy Apparently Due to a Lesion of the Vasomotor Center of the Brain". The syndrome was further elaborated by Kalischer in 1901, by Dmitri in 1923, and finally by Weber in 1929. The description of the pathological changes by Krabbe in 1934 has led his students to urge the addition of his name to the eponym.

The term encephalotrigeminal angiomatosis was introduced recently and would seem a proper name for this anomaly. The findings comprising the full syndrome are: 1. a unilateral hemangioma of the cutaneous distribution of one or more branches of the trigeminal nerve, 2. glaucoma on the same side, 3. an angioma in the leptomeninges, with calcification of the underlying cerebral cortex on the side of the cutaneous hemangioma, 4. mental retardation, and 5. focal seizures and/or contralateral hemiplegia.

The cutaneous portion of the complex is varied. It may appear as a smooth port-wine mark of varying size and prominence or as a large and prominent cavernous or turgid lesion. The may cover the entire cutaneous distribution of one or more branches of the trigeminal nerve or be a small and insignificant hemangioma. Usually the upper two branches of the nerve are involved and include hemangiomatous change in the mucous membrane of the hard palate and the alveolar ridge of the maxilla. Involvement of the eyelid and conjunctival tissue may be prominent or minimal. In some patients the same type of hemangioma has appeared in nerve distribution on an extremity or other portion of the body along with the facial hemangioma.

Like the skin, the eye shows a variety of changes. Glaucoma is the best known finding but colomboma, keratoconus, and heterochromia are also seen. Eye manifestations are striking in some cases, less so in others. Some authors find a parallel between the time of onset and the severity of glaucoma, as well as the general degree of ocular involvement, and the size of the angiomas in the eyelids. Glaucoma is presumed to result from obstruction of the canal of Schlemm by choroidal and uveal vascular engorgement. Some patients show only minor dilatation of retinal vessels; others have diffuse and prominent angiomas of the choroid, retina and uvea. Cupping of the disc is expected with glaucoma but with intense vascular engorgement papilledema has been observed. Apparently glaucoma is not always limited to the side of the hemangioma, but may be bilateral.

Neurological symptoms are often present in infancy and, in most cases, during the first decade of life. Most patients show mental retardation, and many show focal seizures. Hemiplegia is not always present or marked. Electroencephalographic tracings frequently show prominence of dysrhythmia in association with the intracranial angioma, yet other reports indicate prominent and localized cerebral electrical dysfunction in more remote areas apart from the angioma. Still others show diffuse seizural pattern and electroencephalographic aberrations.

Intracranial calcification is a striking change. Roentgenograms of these areas of calcification are characterized by the appearance of waving doubly contoured lines. These were formerly felt to represent the course of vessels within a calcified angioma, until Krabbe showed that the calcification appears predominately in the upper cellular layers of the cortex and not in the overlying angioma. These lines are now thought to represent the contrast between sulci of the cerebral surface and adjacent calcified areas. One might expect areas of calcification to occur in the frontal areas of the hemisphere, but in reality this is quite uncommon. Most areas of calcification and angioma formation are found in the parieto-occipital region of the hemisphere.

The hemangioma is within the leptomeninges, and the dura is rarely involved. Fine calcific granules such as occur in the angioma have been found in the dura. Microscopically, angiomas of the cranium are reminiscent of mixed simple-cavernous hemangiomas of the skin.

Neurosurgical removal of areas of calcification, along with the angioma, has been carried out in some patients, and in such cases the seizures have been abolished. Mental aptitude has not been improved,

however. Other therapy includes roentgen radiation over the cerebral lesion, with which improvement has been reported.

In summary, the cutaneous and ocular findings of this syndrome show the greatest degree of clinical variation. Central nervous system findings can vary and must be expected also. From the external oculocutaneous features, we have no guide to presence of or severity of intracranial involvement, which emphasizes the need of complete evaluation in all suspected cases.

Maffucci Syndrome

This disease complex has attracted interest only in recent years. To date 36 cases have been reported, most of these in the last two decades. The principal features of this syndrome are: 1. multiple enchondromata, 2. progressive dyschondroplasia, and 3. multiple hemangiomas.

The syndrome was described by Maffucci in 1881. His paper appeared near the time of the description of dyschondroplasia by Ollier, and perhaps because of greater interest in the papers of Ollier, the work of Maffucci received little attention.

This disease first appears in children, usually between the ages of 3 and 5 years. The hemangioma and bony change usually appear at about the same time, but with some tendency for bone changes to appear first. The dyschondroplasia does not appear to be unique or separate from the Ollier type. The changes in bone begin with widening of the metaphysis of the long bones and formation of enchondromata near joints. Bony change is progressive up to puberty when it ceases. Most patients show considerable bony deformity by this time.

The hemangiomata are deep in the skin and are in greatest number in the hands and feet. Hemangiomas in and about bones of the hands and feet may contribute to the bony deformity in these areas. With formation of the angioma an increase in temperature of the extremity, increase in hair growth, and increased sweating of the part may be observed. These alterations in temperature and sweat mechanisms suggest possible arteriovenous communications; but such findings have not been reported; nor have studies of oxygen saturation of the blood indicated that such could exist. Dilated veins near these multiple hemangioma have been described by visualization with contrast media. The importance of this finding is not clear.

The occurrence of pain in some cases has led to the conjecture that these hemangiomata represent multiple glomus tumors. There is no

pathological evidence supporting this view, and several authorities deny existence of multiple glomus tumors of such number, size, and pattern.

Only one fatality attributed to this syndrome has been reported. This occurred in a patient who died of chondrosarcoma arising in the region of the sella turcica. At autopsy no primary central nervous system lesions were seen, and no angiomas were reported in parenchymatous organs. Histologically these tumors are similar to the cavernous type of hemangioma. The bone and cartilaginous changes are a dyschondroplasia similar or identical to the Ollier type.

An interesting case was reported in 1956, in which there was lipomatous infiltration of skeletal muscle. If this proves to be a consistent finding in the future, the features of the syndrome may need to be expanded.

There is no explanation for the existence of this peculiar dysplasia. Certainly it cannot be considered a neoplastic process from the present evidence and reports available. The diagnosis should not prove to be too difficult if one but keeps in mind the interesting features of this disease.

Klippel-Trenaunay-Weber Syndrome (Osteohypertrophic vascular nevus)

The eponymic title for this syndrome was derived from the case reports of Klippel and Trenaunay in 1900, and Weber in 1907. The first partial recognition of the syndrome was by Geoffrey-Saint Hilaire who in 1832 described partial gigantism with vascular nevi. The full syndrome was reported by Trelat and Monod in 1869. Klippel and Trenaunay popularized the syndrome on the European continent, and Weber reviewed the disease in the English literature. Up to date there have been about 120 cases of this syndrome reported under 30 different names.

An adequate evaluation of reported cases is impossible because of the lack of uniform terminology and incompleteness of description. The definition of this syndrome has only the limits which each individual author gives it. Klippel and Trenaunay described the syndrome in three parts: 1. unilateral plain (simple) angioma, 2. unilateral congenital hypertrophy of the bone and soft tissue on the involved side, and 3. varices on the same side, appearing at birth or in early youth. Weber, in 1907, in his article "'Haemangiectatic Hypertrophy of Limbs—Congenital Phlebarteriectasia and So-called Congenital Varicose Veins", set up the same criteria.

Classification of the syndrome has been varied, but there are two popular classifications. The first and more widely used is:

1. Complete form.
2. Arrested form.
   a. Nevus type.
   b. Osteo-hypertrophy.
   c. With varices.
3. Abnormal forms.
   a. Alternating type (e.g. both legs affected).
   b. Cross type (e.g. nevus on one side and hypertrophy on the other).

The second classication (by Langsteiner and Stiefler) is based solely on osseous manifestations and includes:

1. Partial hyperplasia (part of a leg).
2. Extremity hyperplasia (mono-hyperplasia).
3. Bilateral hyperplasia.
4. Crossed hyperplasia.
5. Incomplete hemihyperplasia.
6. Complete hemihyperplasia with participation of the internal organs.

We offer no new limits, nor do we urge adoption of either of the foregoing; but rather we wish to discuss the syndrome within the boundaries of the original description. The syndrome has two main features: 1. systematized hemangiomas, and 2. hypertrophy of the involved side.

The vascular findings are varied. The most common is the racemose hemangioma, ("congenital varicosity"), which appears shortly after birth. This usually appears on the same side as hypertrophy, but there are reported instances of its occurrence on the opposite side. Other hemangiomas also occur, with or without the racemose hemangioma, and undoubtedly are part of the syndrome. A bruit is often heard over the racemose hemangioma, depending upon the arterial supply. If there are multiple arteriolar communications no bruit will be heard. These hemangiomas may be present at birth and may give the only clue to the impending development of the complete syndrome.

The osseous manifestations are hypertrophy of the bone on the involved side. The right side is involved twice as often as the left, the lower extremity in 58 per cent, the upper extremity in 22 per cent,

and both upper and lower extremities in 20 per cent. Partial hypertrophy is thus more common than the very rare hemihypertrophy. The long bones are hypertrophic, being normal in configuration, but too big for the size, age, weight, and general bone structure of the person involved. There are cases reported in which the involved bones did not hypertrophy uniformly. Hypertrophy is usually present at birth, or is apparent within a few years. Reported increases in length have been from 1 to 19 cm. As a result of the increase in size, there is often a compensating scoliosis. Hypertrophy is often overlooked in the early stages because a certain amount of asymmetry is normal. Just where this asymmetry becomes abnormal is hard to define. Hypertrophy usually is not noticed in these early stages because the hemangioma which is present distracts the observer's attention from the hypertrophy.

To complicate the problem further, recent authors have described so-called typical cases of Klippel-Trenaunay-Weber syndrome with bone atrophy instead of hypertrophy. These cases fit all the criteria of Klippel-Trenaunay-Weber syndrome except for the atrophy.

There are other cutaneous manifestations which can occur with or without the vascular abnormalities including: 1. *café au lait* spots, 2. pigmented hairy nevus, 3. hypertrichosis, 4. excessive secretion of the sebaceous and sweat glands, 5. thickened hair, 6. abnormal nail growth, 7. vitiligo, and 8. increased temperature of the involved side. These may be the first clues to the diagnosis of the syndrome.

Twenty per cent of the patients are mentally deficient. In a smaller number of patients, there is hypertrophy of the cerebral hemisphere on the involved side. Both contralateral and bilateral cortical hypertrophy have been reported. Hypertrophy of the extremity is not limited to the bone but involves muscle, nerves, and subcutaneous tissue. Other organs which have been described as abnormal in the syndrome are the heart, with hypertrophy and murmurs; the eye, with enlargement of the cornea, pupils, and the osseous orbit; the liver, with hemangiomata; and the viscera, with hypertrophy limited to one-half of the body. In about 50 per cent of the cases of congenital hemihypertrophy other congenital abnormalities are present. Rarely there has been endocrine dysfunction, and in one case there appeared to be a mixture of the Klippel-Trenaunay-Weber syndrome and Sturge-Weber syndrome.

Multiple and unsatisfying theories of causation have been pro-

pounded for this syndrome. Based more on speculation than fact, they include: 1. vascular lesions, 2. lesions of the central nervous system, 3. endocrine factors, 4. embryonic defects of the vegetative nervous sytem, 5. mechanical influences in utero, 6. heredity, 7. embryonic variants with an atypical form of twinning in the very earliest division of the fertilized ovum, and 8. combinations of the above.

Therapy is confined to the treatment of the complications of the various parts of the syndrome which cause symptoms.

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Special Article

Roundsmanship

Henry Jacob Bulfinch, '56

They think that they shall be heard for their much speaking.
(Matthew, VI, 7)

Stephen Potter has done great service to civilization by defining certain principles of conduct which he has identified as Lifemanship.[1] This is best illustrated by its fundamental axiom, "if you're not one up, you're one down." Perhaps nowhere is Lifemanship better found than at what at inside* hospitals is called Grand Rounds.

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Reflection on the term "Rounds" confirms one's faith in the power of the English language to bewilder. What formerly was an occasion upon which a group of doctors walked together around the wards dispensing knowledge and healing suggestions has become a weekly carnival having for each who attends some special significance. For some, particularly the nurses, it is a refuge from the hurly-burly of their duties; for others, an educational exercise both to give and to receive. For many it is a chance to sit back** and enjoy the passing scene in which the Houseman, the Rowman, and—at times—the Patient, figure so prominently. It is the Housemen and the Rowmen who create the atmosphere of Rounds known to Galen, that Master Roundsman, as the "Aura Roundsealis", and it is to them, that the principles of Roundsmanship apply.

Between the two, the Houseman and the Rowman, there exists a

* To distinguish them from "outside" hospitals.
** Or, in some cases, to sit hunched forward, head in hands.

struggle that is Martian in scope. The Houseman, dressed in white as befits his tender years, is the champion of trial and error. To him is allotted the first move. The Rowman, by contrast, is on the side of accumulated wisdom and clinical experience. To a certain extent he is at the mercy of the Houseman but even more, he is at the mercy of the other Rowmen who occupy the rows or benches in definite order: Frontrowmen, Secondrowmen, and Backrowmen.

I. HOUSEMANSHIP

Since it is the Houseman who arranges the events of the carnival, we must first direct our attention to the rules and general principles which govern his conduct, not overlooking certain pitfalls and grave errors into which he may fall.

Since the Houseman knows what the events are to be, he has little interest in the show itself other than to practice Housemanship in order that when the hour ends he will be one up and the Rowmen one down. Ordinarily he will do well to see to it that so far as he is concerned the one-downess is not confined to any one Rowman but to all the Rowmen as a group. There are many ways to accomplish this which for convenience we will divide into three categories, Houseman-Rowman play, Houseman-Patient play, and the Proper use of Props.

A. Houseman-Rowman play

Since the Houseman makes his first contact with the Rowmen when he begins to present the case at hand, the traditional opening gambit is to give the patient's chart to the Frontrowman who holds the lowest position of those present, rather than to the senior man. This does not necessarily annoy the senior man, but is most effective in irritating the other Frontrowman because protocol has been so hideously violated. If a Secondrowman has by some indiscretion taken a position in the front row, or if he has been trapped there from a previous round, it is a most effective play to hand the chart to him.

Another unfailing way for the Houseman to score is to present a case in which treatment has been unsuccessful or some unforseen dire complication has supervened. This should be reported in a matter-of-fact, rather diffident tone of voice, a "bad-luck-but-there-it-is" sort of attitude. The same situation may also be dealt with by assuming a super-professional, calm demeanor, talking down to Rowmen in much the same manner in which the anxiety of the patient's family was allayed on the previous day.

This is a potentially disastrous play, however, which may well leave

the Houseman "one down" if the Frontrow is united in its horror of the direct violation of time-honored custom. Wise is the Houseman who splits the Opposition by injecting into his presentation a non-sequitur about which there is an emotional rather than a rational disagreement.

In Housemanship the element of mystery must never be neglected. The point of the presentation and the nature of the problem should be suitably obscure. The advanced Houseman can bring the Rowman to a point of frustration at which he will attempt to elicit the essential information by questions that he will hardly dare ask for fear that the information has in fact been given out but that due to his inattention he has missed it. To ask for it now would make him lose face. Whether he asks for it or not, it is good Housemanship to have created the conflict.

Much can be made of presenting a patient *en surprise,* that is, failing to notify the Rowman who has operated upon the patient that his handiwork is to be on display. The Rowman will, therefore, have no prepared remarks and will not, it is hoped, have had time to look up the one significant article on the subject. He will thus be torn within himself as to how much to say about the case. Conflict again will have been created. The situation can be further exploited by the Houseman if he makes a point of removing the appropriate volume from the library several days before and perhaps even reading from it after the Rowman has stammered out his garbled and ineffectual words.

In the same vein, a situation of strength can be created by inviting a member of the Medical Staff to discuss a case with a prepared speech and slides for perhaps thirty minutes of an hour in which the showing of six cases had been planned. Though this may contribute greatly to the education of the representatives of the Nursing School and Medical Arts Department seeded among the Backrowmen, it produces little but a restless ferment among those down front.

B. Houseman-Patient play

The trained Houseman becomes expert in his handling of the patient. Nothing, for example, so breaks the ice as the presentation of an infant surrounded by all the trappings of childhood; balloons, stuffed animals and the like. On such occasions the close observer will be mildly startled to see several of the Frontrowmen surreptitiously trying to win the attention of the tiny patient who according to time-honored custom is presented as "Mister" or "Miss" Bizbee. At the

other end of the scale, a spry elderly patient should be introduced as of something greater than her actual age. This will be immediately corrected by the patient and if the Houseman can then manage to blush prettily, he has scored again. By constant, it is distinctly poor play to ask the patient how he or she feels since only one reply is acceptable.

Many broad points of policy must be decided in the handling of patients. Much can be said for rushing the bed in and out so fast that no one can be sure whether or not it actually contained a patient. By contrast, the patient may be kept throughout a long and complicated presentation, during which preferably he should lie curled up in a ball, back to audience, with face covered. A particularly effective form of Housemanship is to present three to five patients at once, so that the amphitheatre furnishes a scene of intense activity not dissimilar to a Bruegel canvas.

Bed crashing is best reserved for patients who are no more than one day post-operative. As the bed is run solidly against the door jamb, a barely audible murmur of "shame, shame" can be made out running through the audience. If the bed in question is equipped with a five-pint bottle hanging from its side, the effect is heightened. A particularly useful maneuver is to arrange that a constant drainage catheter or common-duct tube should drain not into the bottle, but onto the floor during the presentaion of the case.

C. The proper use of props

Aside from the patient and his various appurtenances, the Houseman has at his disposal only a few props, chief among which is the x-ray viewing box. With proper use, however, much can be made of this. It is axiomatic, for example, that at some time during the rounds films should be displayed in a reversed position—so called *situs inversus radiologicus.* The combinations possible with spotfilms are inexhaustible. A particularly useful maneuver is to drag the viewing screen forward so that all may inspect the films more closely. At the critical moment the light cord to the appartus becomes inadequate and all is dark. Great effect can then be created in the efforts to restore illumination.

Much can be done by the proper use of the pathological specimen. During the presentation of the patient, it should be kept in a prominent place discretely covered by a voluminous pile of wet rags or sodden paper towels. It should always repose upon an evil-looking en-

ameled platter or in a battered and obviously contaminated basin. As the patient is wheeled out, the trophy is then triumphantly unveiled, quickly thrust upon the nearest Frontrowman with the suggestion that it be circulated freely so that all may share its contagion. So far as possible it should be arranged that the pathologist has taken sufficient sections from the key point of the specimen so as to make orientation virtually impossible.

II. Frontrowmanship

The Frontrowman occupies a key position in the structure of Rounds. His place is more than to sit in judgment, he represents continuity and accumulated wisdom. This is to be scattered among the pilgrims who have come to learn. At the same time he must defend his place among his peers and guard himself from the Houseman. His techniques are many.

The authority and stature of a Frontrowman is often demonstrated by the degree with which he undertakes to converse with his neighbor while a case is being presented or another individual is discussing it. In effect, he creates a diversion. If this is aimed at the Houseman, it can be made most effective by inclining one's head towards a neighbor, looking the Houseman directly in the eye and asking in a stage whisper, "What's this boy's name?" The answer, "Don't know—must be a striker," virtually assures triumph.

A very useful form of Diversionship is to whisper to one's neighbor in tones inaudible even to him some witticism of any quality, followed immediately by a loud chortle or scarcely subdued laugh. The neighbor can only nod his head vigorously and smile wanly. The impression, however, will have been created among the Housemen and Rowmen alike that some priceless thing has been said or done which they in their dull-witted or inattentive state have failed to appreciate.

There is the technique for capturing the center of the verbal stage by the judicious interruption. This requires finesse that comes of years of experience and should be done in a way that the other members of the audience relish. Perhaps the surest method is that of the interrogative sentence. This is a question which during its first few words sounds as if the asker were really requesting information. A deft Frontrowman, however, can easily, without drawing breath, allow the words to reorient themselves so that instead of asking, they tell. Thus, if a patient has had necrosis of a colonic suture line following resection, one can say, "Knowing very little about these matters, I should

like to ask whether you don't think it is wise to examine the bowel near the resection edge for pulsating vessels?" One thereby creates the effect that (1) he knows a great deal about these matters; (2) that with razor sharpness he has come straight to the heart of the matter.

No positive statement on the part of a Houseman or Secondrowman should be allowed to pass without some qualification by a Frontrowman. This can often best be done by citing from one's own experience examples of somewhat similar cases which really have little bearing on the subject at all. For instance, if a patient is presented as an example of the benefits of a given treatment A, the deft Frontrowman will not discuss treatment A (with which indeed he has had no experience), but merely describe with warmth and humor a case which he has handled with brilliant effect by treatment B, the method long established as the best, although not always adequate, one.

Complete denial of a positive statement made by a Junior is best handled by Distortionship. After a judicious interruption, the Frontrowman proceeds sonorously down the verbal stage with a definite sequence of moves: (1) expression of great interest in what Doctor Doe has said; (2) direct misstatement of Doctor Doe's argument prefaced by "If I understand you correctly"; (3) entire agreement with the revised, mirror-image version of Dr. Doe's argument. An authoritative impression of good fellowship is thus created except in Dr. Doe, whose meaning having been exactly reversed by Dr. Frontrowman, realizes that he has been had again.

The courageous and righteous castigation of the Houseman for some minor error in the handling of a case is always effective in creating an impression though it can only be done by One Who Has Arrived. The Frontrowman, however, must be continually *en garde* lest he is being mouse-trapped into a display of platitudes. A method of correction that invariably succeeds is to read from the record any significant item which, in his presentation, the Houseman has failed to incorporate, or has quoted incorrectly. Much can be gained by an obviously careful perusal of a paient's new, or old, record, with on occasional aside to one's bench-mate. Unless the Houseman is exceedingly well briefed in the case at hand, or has taken the precaution to see that the record has not fallen into the hands of a known Record-Reader (see Sec. A, Part I, Housemanship) he can usually be put completely off balance.

Humility has many uses to the Frontrowman. Should a Backrowman ask a question that defies answer, the alert Frontrowman will hesi-

tate, verbally, to reply to the question. Then, while pleading *nolo,* he will nail another Frontrowman by tossing the ball directly in his lap, thus: "Perhaps Doctor Throop would be willing to answer that question since he knows more about these matters than I." Dr. Throop, of course, does not, but feels impelled to say something. This thoroughly discredits him for the balance of the Rounds and Frontrowman A emerges as the humble handservant of Truth. The Backrowman is more confused than ever and wishes that he had not asked the question. The whole episode serves to discourage further questions from the Backrowmen and thus a proper Aura Roundsealis is preserved.

A successful gambit may be occasionally achieved by complimenting the Houseman and his associates effusively on the management of a case. This is particularly effective if the patient really represents a rather mediocre result of what is, at best, a questionable form of treatment. The Frontrowman should purposely misinterpret the result to be an excellent one. This ostensibly magnanimous maneuver creates a sense of uneasiness in the Houseman who knows the result is not a good one, knows that the Frontrowman knows, and cannot understand his motive in using the Patronship play.

There is one prestige maneuver, namely Camaraderieship, that is of great utility. In essence, Frontrowman A, finding himself cornered in argument with colleague B, quotes as his authority leading figures in other medical capitals whom he refers to by their nicknames, thus: "When I was last in London, Archie Heneage told me that he had given up cutting the stem cryptoleus and relies entirely upon drainage through the ptyaloid apparatus." The drawback to this form of play is obvious. Frontrowman B, having been given the green light, may be able to quote celebrities faster than A. If play is then conducted along these lines, much prestige may be lost by both A and B as nicknames fly back and forth like pillows in a pillow fight. A surer form of Camaraderieship is to introduce a member of the international set to the front row. If this figure can then be lured into making a few suitably obscure remarks or will, at the appropriate moments, nod and grunt knowingly, his sponsor has indeed scored.

III. SECONDROWMANSHIP

The Secondrowman may be considered a larval stage. He will fall into one of three categories: assistant surgeons of the regular staff, middle-aged regular attendants at Rounds who possess considerable stature

in their own "outside" community, and Fourthfloormen.* The Secondrowman operates according to the general rules of order laid down for his seniors. Thus he may create a diversion, interrupt, qualify, distort, be humble or expound.

The Secondrowman ordinarily should be humble, with one exception to be noted later. Thus he should apologize for the presentation of one of his cases in some such manner as: "I thought at the last minute that it would be worthwhile to bring this patient down since he perhaps illustrates one or two interesting points." This creates the impression that in the rush of practice he has been able to gather his wits long enough to recognize a problem when he sees one, but not long enough to prepare a discussion of it. The discussion which follows must be carefully given in a halting and hesitating manner so as not to betray the two hours of rehearsal which had been secretly devoted to it the night before.

The Secondrowman, middle-aged-regular-attendant type, is expected to look pleasant and relaxed and to say very little. He should, however, fill in the awkward gaps in the proceedings which occasionally occur, by telling of an interesting case with which he has just been confronted. This should never be done with an air that would lead anyone to suspect that such cases are not often seen in this large teaching center. It should be accompanied by an earnest attitude as if seeking guidance and advice. Since no advice will be forthcoming the remarks should be brief.

The Fourthfloorman's contribution to Rounds should be preserved as a very special one. He has come to deliver a Message and the Message will be good. He should come to Rounds early and be discovered checking formulae that he has put upon the board by doing quick calculations in the corner using Greek letters and a slide rule. A bit of unusual apparatus which blinks balefully and which emits ominous and cosmic ticking sounds can create a superb atmosphere of suspense and anticipation. The Message must be a crisp one. It need not instruct. It need not be clear. But it must be authoritative. Camaraderieship is usually out of place, but there should be implied a complete familiarity with and mild contempt for other prominent workers in the field, their co-workers and their laboratories.

If the Message has been delivered correctly, there will and should be

* It is recognized that this term may be a local one and perhaps Researchman, or even, as some have suggested, Trainedsealman, might be more appropriate. However, the "Fourth Floor" is so distinctive a place, the author has chosen to preserve this terminology.

no discussion. An admiring silence will ensue, broken only by the Houseman jouncing in the next patient, who, if the Fourthfloorman has arranged things properly, should represent the most banal of problems such as a case of appendicitis treated by appendectomy.

IV. BACKROWMANSHIP

Backrowmanship, as has been implied above, is merely the art of sitting still.

REFERENCE

1. Potter, S.: *Lifemanship*, Henry Holt & Co., New York, 1950.

EDITOR'S NOTE: Bulfinch is to be warmly congratulated on the publication of this work, for he has indeed performed a service for Medicine in general and in particular for academic or Halls of Ivy Medicine. With most of his concepts we are in full accord. Although its fundamental principle, i.e. if you're not one up, you're one down, remains constant, we are sure that the actual practice of Roundsmanship varies from one locality to the next. We hope that it will not appear presumptuous to expand somewhat on Bulfinch's theme.

Ever since our first introduction to the memorable works of Stephen Potter[1,2], we have been convinced that it is in a medical setting that Lifemanship really comes to fruition. The halls and classrooms of a teaching hospital spawn Lifemen as a swamp does mosquitoes. We might profitably explore some further ploys and gambits in the practice of Medical Lifemanship—or Roundsmanship as Bulfinch has so aptly called it.

Osler-quotesmanship. This relatively simple ploy can be put to use either by the beginning Secondrowman or by the Backrowman. (As we shall develop presently, we do not agree with Bulfinch on the role of the Backrowman.) It consists merely of stating (Secondrowman): "As Sir William Osler once said, '_____.'," or by asking (Backrowman): "Wasn't it Sir William Osler who said, '_____.'?" This, of course, not only acknowledges once again Medicine's debt to a great physician and teacher, but when appropriately used implies that the speaker is *well-steeped in medical classics.* Suitable quotations are readily available in "Aequanimitas with Other Addresses,"[3] a volume thoughtfully supplied to graduating medical students by one of the pharmaceutical houses. However, a Secondrowman must be careful to use this ploy only in discussions with persons considerably less sophisticated medically than himself, preferably second or third year students. Backrowmen may utilize it at will, since even

a faint glimmer of acquaintance with medical history exhibited by a member of this group will be roundly applauded.

Advanced Osler-quotesmanship. In contrast to the preceding, this ploy is a favorite of some of the more senior Secondrowman and at times can stand the Frontrowman in good stead. It is especially helpful, for example, if Frontrowman A is being badly outquoted on subjects in the current literature by his fellow Frontrowmen and perhaps —perish the thought!—even by the Secondrowmen. If, in such a circumstance, the harried Frontrowman can manage a philosophical quotation beginning, "As Sir William Osler said, *while at Pennsylvania,* '_____.'," the discussion can usually be terminated effectively and in favor of Frontrowman A. Naturally, this ploy carries with it the implications of the preceding one and the qualifying phrase, *while at Pennsylvania,* establishes the speaker as a *true follower.* It must, however, be used with caution for there is always the danger that Frontrowman B will know that the quotation in question stems from the Oxford era.

Camaraderieship, Continental Type or the Deutschland über Alles Gambit. Bulfinch has called attention, and rightly so, to the camaraderieship maneuver. However, in our opinion the effectiveness of this is increased manyfold if combined with the Macintosh Finisher or Advanced Languaging as described by Potter[1]. The play then goes something like this: Frontrowman A has just finished examining a patient with a most confusing array of symptoms and physical findings. A spirited discussion, generating more heat than light, is taking place among the Secondrowmen as to whether the patient has Addison's disease, non-tropical sprue, pan-hypoputuitarism, or the Rénon-Delille syndrome. At this point Frontrowman A says, "As Prof. Otto von Dieselmacher of Leipzig used to say when confronted by a problem of this type, 'Wenn man eine Gesellschaftbrachen hat, dann man die alte Meidung wachst.'" Adjournment of the conference regularly follows immediately. It is believed that each year at least two third-year students decide to drop out of school after witnessing the appropriate application of this gambit.

The Subspecialty Denial. Held in high regard especially by Secondrowmen, this gambit boasts a wide field of usefulness. Secondrowman Z, whose interest in, say, hematology is well known and whose relative naivete in the field of cardiology is likewise acknowledged, is confronted with a cardiac patient while making general medical rounds.

After making his examination, he states, "I'm no cardiologist, but I believe this patient has an auricular septal defect." The principal virtue of this gambit is its absolute safety. Possible results are as follows: 1. If subsequent studies prove him wrong, nothing has been lost because, after all, he is no cardiologist, as everyone knows, and at least he has *been thinking* and *willing to entertain a new idea.* 2. Should he prove to be correct he has demonstrated (a.) that he is after all a pretty sharp cardiologist even though he does not consider himself one, and (b.) that cardiology is, in reality, mere child's play.

The Tangential Specific. This should be reserved for the use of Frontrowmen of *real stature* who are expected to comment on every case, regardless of type. The play is as follows:

> Moderator: Dr. Feep, would you comment on this interesting case of multiple myeloma?
>
> Dr. Feep: The thing that has always interested me about this disease is the renal involvement that often occurs. The best classification of renal disease with which I am familiar is the one proposed by Ellis in the British Medical Journal in 1940, I believe. In it, he divides . . .

The particular conference, ostensibly a hematology case presentation, will of course be remembered as Dr. Feep's clinic on renal disease.

Backrowmanship. It is on this point that we disagree with Bulfinch. We believe that he has accorded the Backrowman rather cavalier treatment. The Backrowman has at his disposal an excellent ploy, Fingernail Clipmanship. The steady snip-snip-snip of the Backrowman's fingernail clipper can effectively dominate a conference. We have seen an able Frontrowman overlook completely a classical machinery murmur simply because an adept Clipman among the Backrowmen provided sufficient distraction. This ploy is, of course, somewhat limited by the relatively small amount of raw material available to the individual Clipman. It is considered poor Clipmanship form to remove one's shoes and socks in an attempt to augment the raw material supply.

Once again we commend Bulfinch on this well-conceived and well-executed study. We believe that his paper is in the nature of a preliminary report. Further study of the problem is warranted, and we await eagerly the report of his follow-up observations.

REFERENCES

1. Potter, S.: *Lifemanship,* Henry Holt & Co., New York, 1950.
2. Potter, S.: *Gamesmanship,* Henry Holt & Co., New York, 1950.
3. Osler, Sir William.: *Aequanimitas with Other Addresses,* Blakiston, Phila., 1943.

Alumni Association

Alumni News

DR. MARTIN S. BUEHLER, '38, is Clinical Assistant Professor of Medicine at the Southwestern Medical School of the University of Texas in Dallas. He has served as President of the Texas Academy of Internal Medicine and is President-Elect of the Texas Geriatrics Society.

DR. ELSA PROEHL PAULSEN, '54, formerly a Fellow in the Department of Pediatrics at the University of Minnesota, is now a Resident in Pediatrics at the Bronx Municipal Hospital Center.

DR. IRVING J. GLASSBERG, '34, has recently returned to Minneapolis to practice after spending many years in New Orleans where he was Chief of Urology at the Touro Infirmary and Clinical Assistant Professor of Urology at Louisiana State University Medical School.

DR. CLYDE E. GRAY, '03, Tacoma, Washington, recently received the 50 Year Gold Pin of the Washington State Medical Association, recognizing a half-century of medical practice in that state.

In Memoriam

DR. PHYLLIS W. BROWN, '53, Fullerton, California
DR. E. K. E. FRITZELL, '30, Grand Forks, North Dakota
DR. JOHN B. HOLST, '95, Little Falls, Minnesota
DR. OSWALD M. JUSTICE, '97, Van Nuys, California
DR. HENRY J. WELLES, '01, Minneapolis, Minnesota
DR. FRED L. BREGEL, '18, St. James, Minnesota
DR. JACOB S. SAGEL, '26, Gary, Indiana
DR. ANDREW A. LOVE, '29, Burbank, California
DR. LAMBERT M. LUNDMARK, '04, Ladysmith, Wisconsin
DR. IDA A. MAC KEEN, '04, Minneapolis, Minnesota
DR. ANNA H. McCLUNG, '99, Pattonsburg, Missouri
DR. HAROLD E. MORRISON, '20, Atascadero, California
DR. GERHARD NESSE, '41, Ephrata, Washington

Medical School Activities

Dr. Bittner Honored

DR. JOHN J. BITTNER, *Professor and Director,* Division of Cancer Biology, was selected as the recipient of the 1957 Bertner Foundation award, which is presented annually by the University of Texas M.D. Anderson Hospital and Tumor Institute in Houston. The award was made to Dr. Bittner in Houston on March 8 at a banquet following which he presented the Bertner Foundation Lecture before participants and guests of Anderson Hospital's Eleventh Annual Symposium on Fundamental Cancer Research. Title of his lecture was "Studies on Mammary Cancer in Mice and Their Implications for the Human Problem." Established in 1950 in honor of the late Dr. E. W. Bertner, first Acting Director of the University of Texas M. D. Anderson Hospital and first President of the Texas Medical Center, the award is presented annually for outstanding contributions to the field of cancer research. We are pleased to offer our warmest congratulations to Dr. Bittner upon receiving this richly deserved award.

Faculty News

DR. DAVID GLICK, *Professor,* Department of Physiological Chemistry, and *Director,* Histochemistry Laboratory, gave lectures on "Some Recent Advances in Quantitative Histochemistry" and "Technical Developments in Quantitative Histochemistry" on February 20 and 21 at the Ohio State University at the invitation of the Anatomy Department. He also lectured at the Biological Warfare Laboratories, Fort Detrick, Maryland, on February 26.

DR. WILLIAM C. BERNSTEIN, *Clinical Associate Professor,* Division of Proctology, will deliver the Annual A. B. Levin Lecture at Louisiana State University on April 24, 1957. He will speak on "The Early Diagnosis of Lesions of the Large Intestine."

DOCTORS N. L. GAULT, JR., *Assistant Dean,* FREDERIC J. KOTTKE, *Professor and Head,* Department of Physical Medicine and Rehabilitation, GLENN GULLICKSON, JR., *Instructor,* Department of Physical Medicine and Rehabilitation, and *Assistant Director,* Rehabilitation Center, and WILLIAM FLEESON, *Medical Fellow,* Division of Psychiatry,

attended the Rehabilitation-Teaching Conference of the National Foundation for Infantile Paralysis which was held in Bandera, Texas, from February 24 to 26.

In Memoriam

Members of the Faculty were saddened to learn of the recent death of DR. BYRL R. KIRKLIN, *Professor Emeritus* of Radiology in the Mayo Foundation and retired Head of the Section of Diagnostic Roentgenology at the Mayo Clinic.

A graduate of Indiana University Medical School, Dr. Kirklin received his postgraduate training in radiology at the Mayo Clinic and at Cook County Hospital in Chicago. An acknowledged leader in the field of radiology, Dr. Kirklin served terms as Chairman of the Section on Radiology of the American Medical Association and as President of the American Roentgen Ray Society. During World War II he was Chief Consultant in Radiology to the Office of the Surgeon General of the U. S. Army. He also served as Secretary of the Advisory Board of Medical Specialties.

To surviving members of his family we join in extending most sincere sympathy.

Postgraduate Education

Trauma for General Physicians

The University of Minnesota, the Minnesota Academy of General Practice, and the Minnesota Committee on Trauma of the American College of Surgeons will join in sponsoring a continuation course in Trauma on Saturday, April 6. The one-day program will be held in the Mayo Memorial Auditorium on the University of Minnesota Campus. This year's course will deal with injuries of the upper extremity and of the spine. Management will be stressed throughout. This is the second in a proposed series of one-day courses, to be held annually, on various aspects of trauma.

Allergy and Chest Diseases

The University of Minnesota will present a continuation course in Allergy and Chest Diseases for General Physicians at the Center for Continuation Study from April 11 to 13, 1957. Management of common problems in these fields will be stressed. Guest speaker will be Dr. Chauncey D. Leake, Associate Dean, The Ohio State University College of Medicine, Columbus, who will also deliver the annual Journal-Lancet Lecture on Thursday evening, April 11. The remainder of the faculty for the course will include members of the faculties of the University of Minnesota Medical School and the Mayo Foundation.

Notice

All continuation courses presented by the University of Minnesota are approved for formal postgraduate credit by the American Academy of General Practice. Attendance certificates will be furnished on request.

Further information concerning the above programs or others to be presented may be obtained by writing to Dr. Robert B. Howard, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14.

Coming Events

April 6 ---------- Continuation Course in Trauma for General Physicians

April 8-10 ---------- Continuation Course in Radiology for General Physicians

April 11-13 ---------- Continuation Course in Allergy and Chest Diseases for General Physicians

April 11 ---------- JOURNAL-LANCET LECTURE; "Drugs Affecting Human Behavior;" *Dr. Chauncey D. Leake,* Dean, Ohio State University College of Medicine, Columbus, Ohio; Mayo Memorial Auditorium; 8:00 p.m.

April 16 ---------- Minnesota Pathological Society Lecture

April 23 ---------- S.A.M.A. LECTURE; "Tristram Shandy;" *Dr. John L. McKelvey,* Professor and Head, Department of Obstetrics and Gynecology, University of Minnesota Medical School; Room 125 Mayo Memorial; 8:00 p.m.

May 6-10 ---------- Continuation Course: Introduction to Electrocardiography for General Physicians

May 7 ---------- DULUTH CLINIC LECTURE; "Studies in Insulin Antagonism;" *Dr. DeWitt Stetten, Jr.,* Associate Director in Charge of Research, NIAMD, Department of Health, Education and Welfare, U. S. Public Health Service; Mayo Memorial Auditorium; 8:00 p.m.

May 13-17 ---------- Continuation Course in Proctology for General Physicians

WEEKLY CONFERENCES OF GENERAL INTEREST

Physicians Welcome

Monday,	9:00 to 10:50 A.M.	OBSTETRICS AND GYNECOLOGY Old Nursery, Station 57 University Hospitals
	12:30 to 1:30 P.M.	PHYSIOLOGY- PHYSIOLOGICAL CHEMISTRY 214 Millard Hall
	4:00 to 6:00 P.M.	ANESTHESIOLOGY Classroom 100 Mayo Memorial
Tuesday,	12:30 to 1:20 P.M.	PATHOLOGY 104 Jackson Hall
Friday,	7:45 to 9:00 A.M.	PEDIATRICS McQuarrie Pediatric Library, 1450 Mayo Memorial
	8:00 to 10:00 A.M.	NEUROLOGY Station 50, University Hospitals
	9:00 to 10:00 A.M.	MEDICINE Todd Amphitheater, University Hospitals
	1:30 to 2:30 P.M.	DERMATOLOGY Eustis Amphitheater, University Hospitals
Saturday,	7:45 to 9:00 A.M.	ORTHOPEDICS Powell Hall Amphitheater
	9:15 to 11:30 A.M.	SURGERY Todd Amphitheater, University Hospitals

For detailed information concerning all conferences, seminars and ward rounds at University Hospitals, Ancker Hospital, Minneapolis General Hospital and the Minneapolis Veterans Administration Hospital, write to the Editor of the BULLETIN, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14.