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# The Antiseptic

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# The Antiseptic

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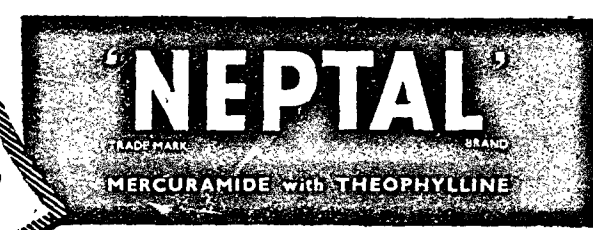
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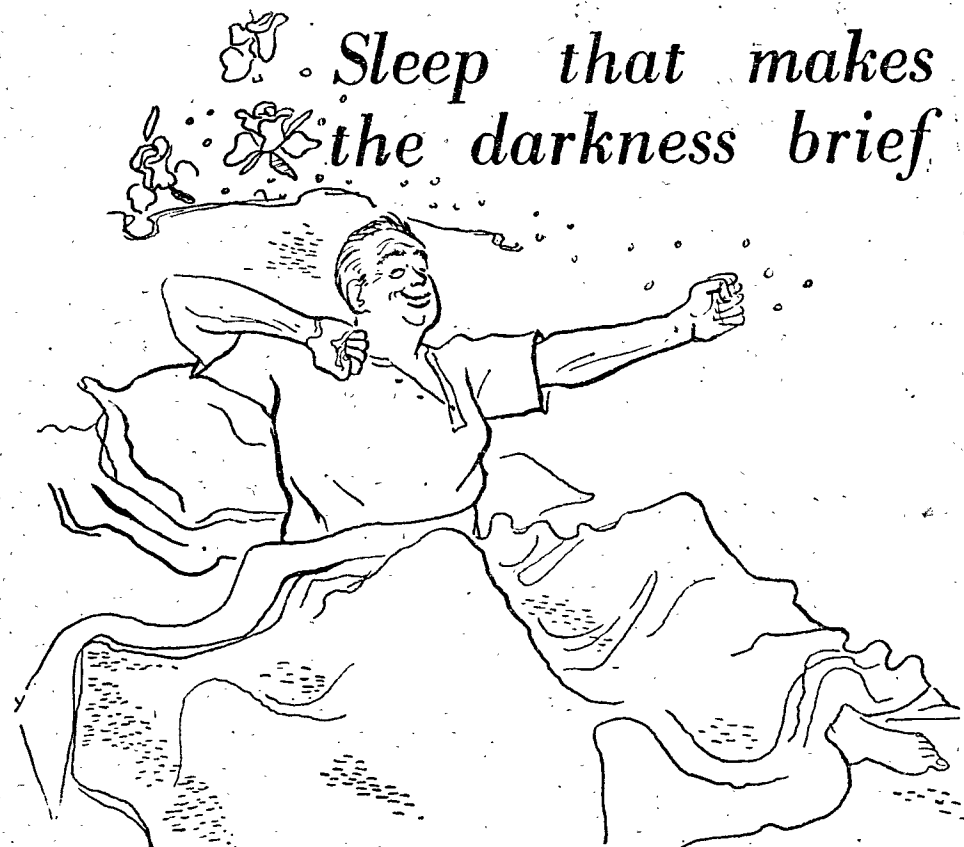
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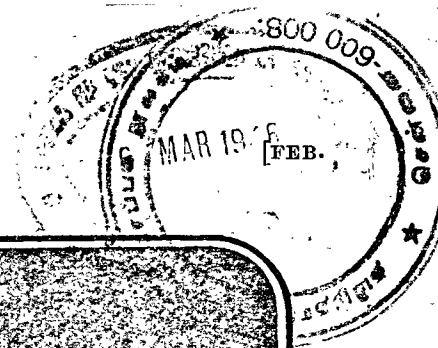
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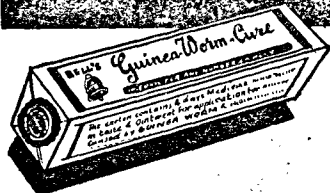
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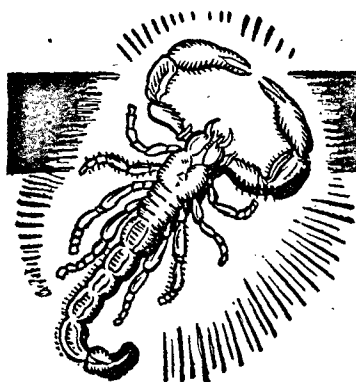
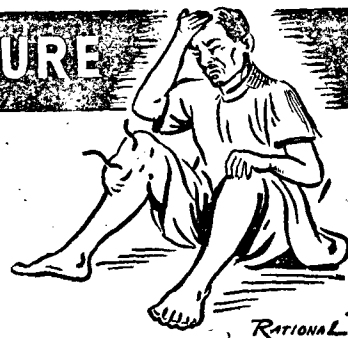
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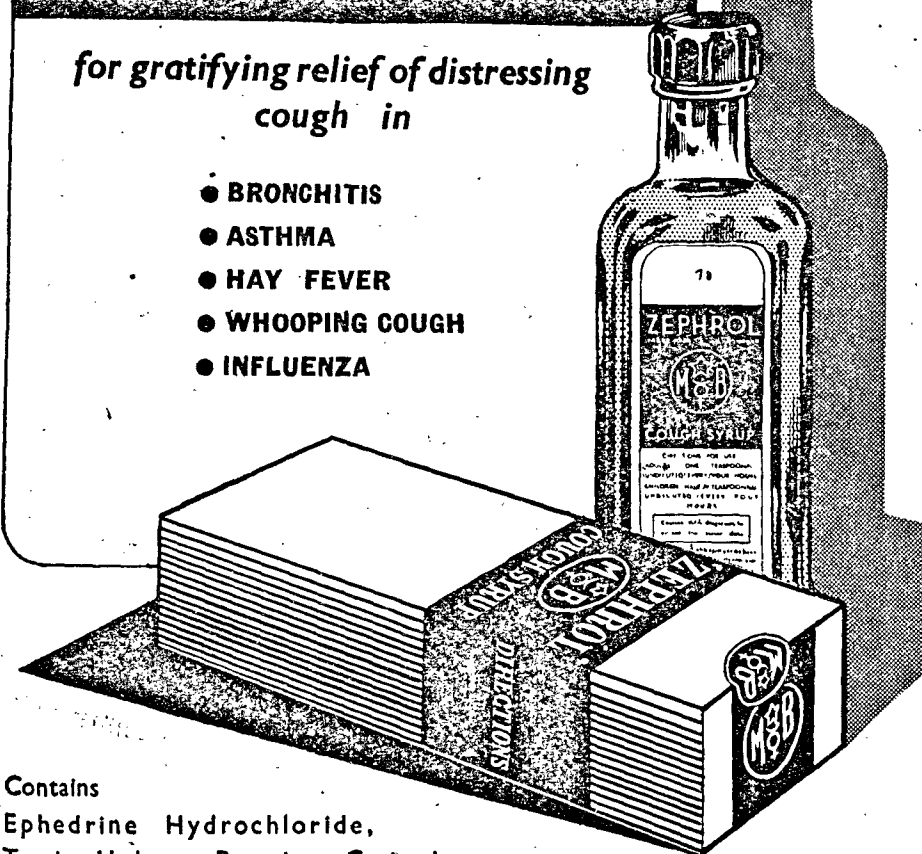
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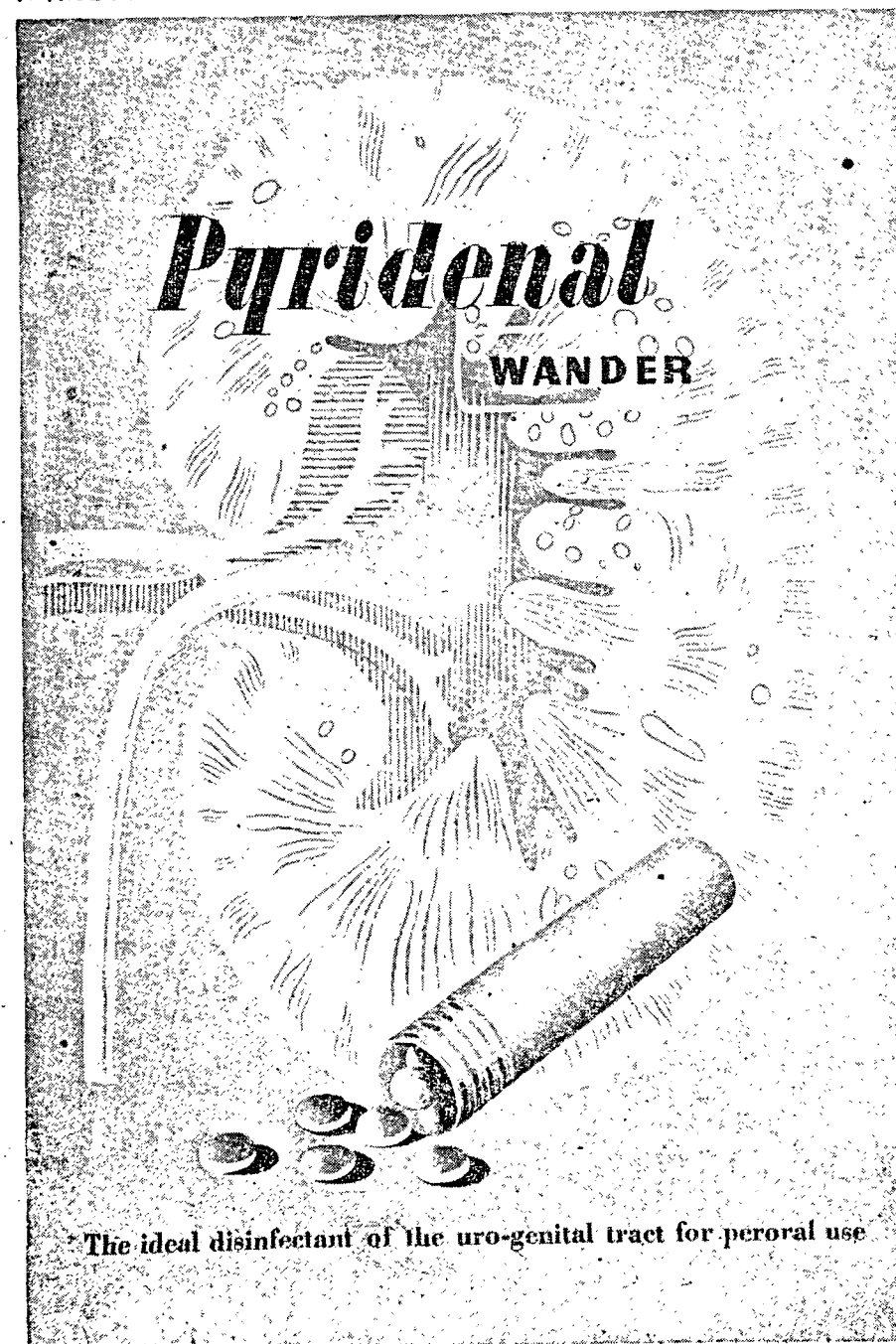
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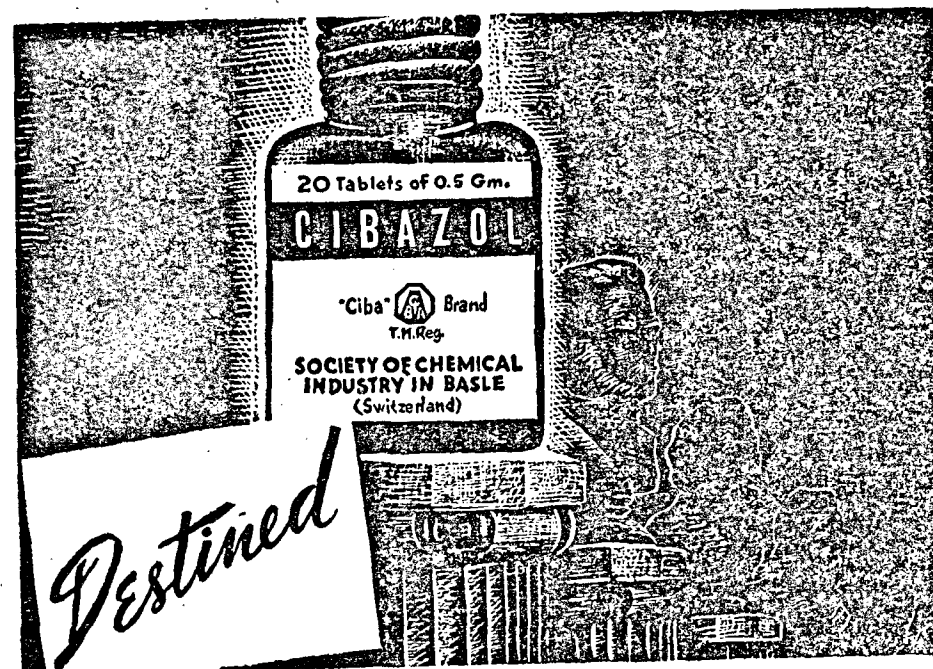
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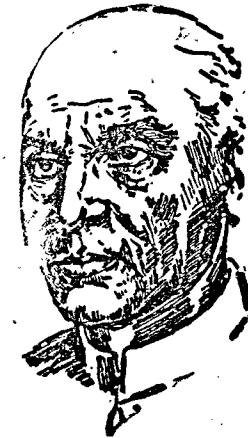
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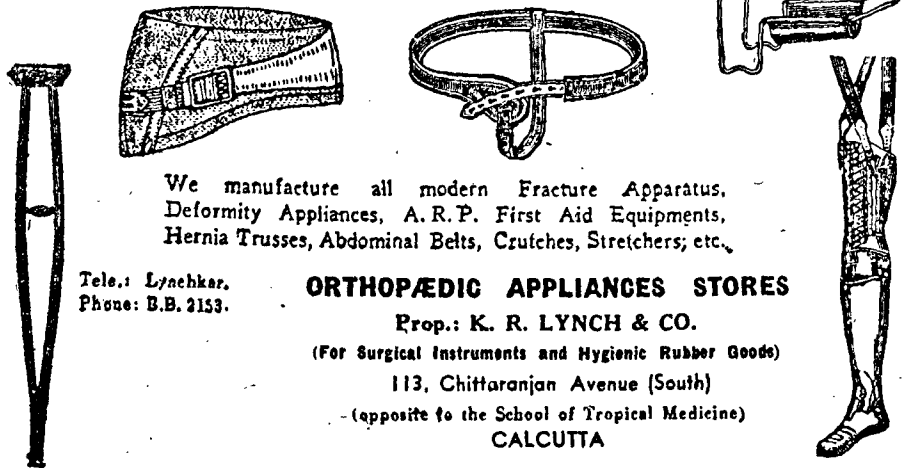
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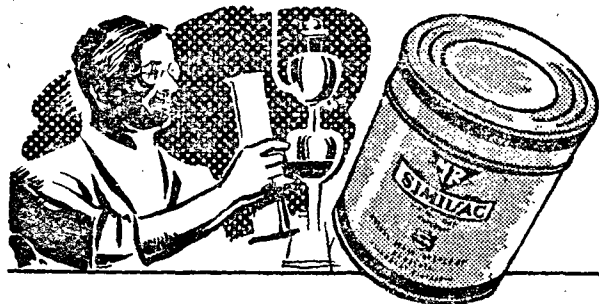
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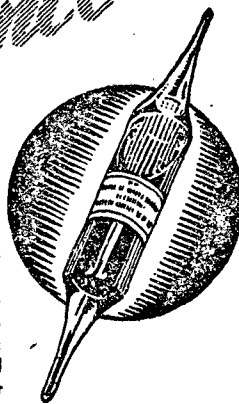
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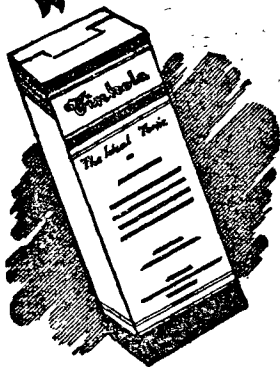
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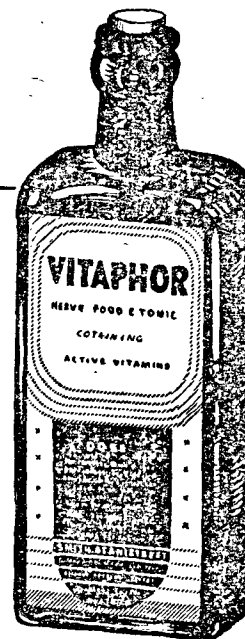
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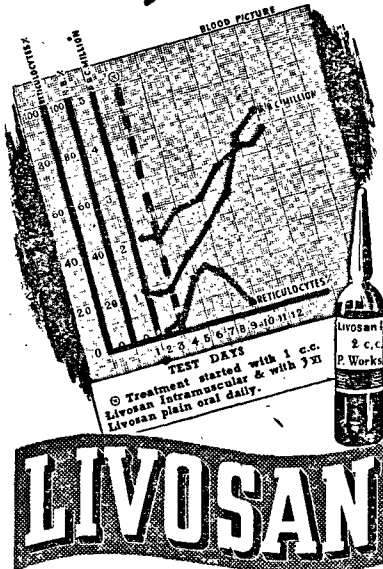
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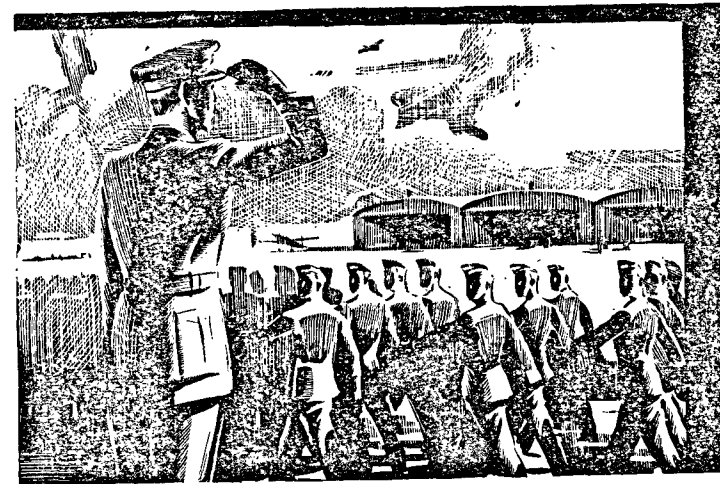
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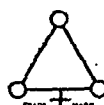
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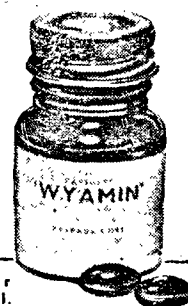
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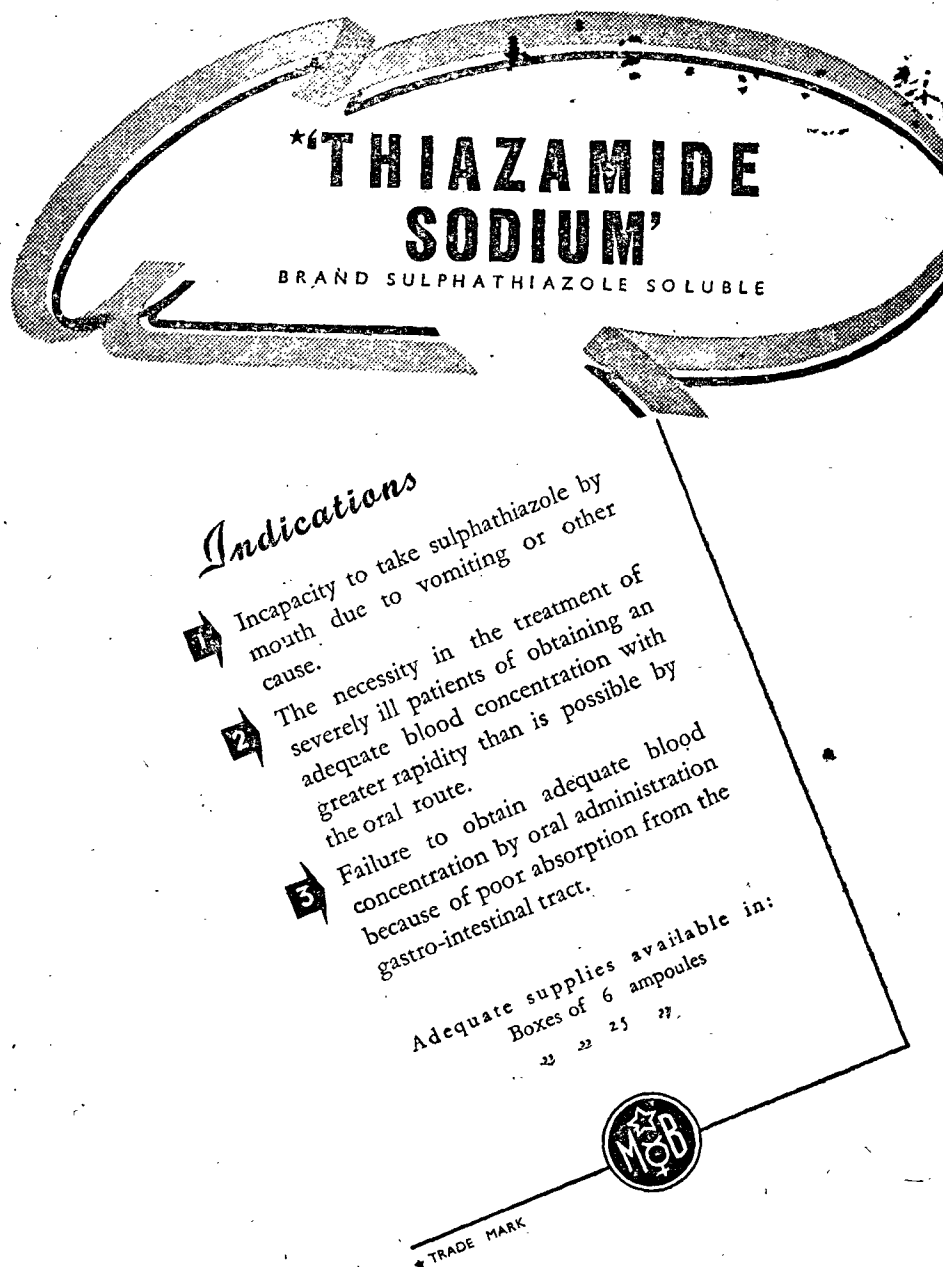
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## Original Articles

### Clinical Aspects of Cardiac Murmurs\*

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A CARDIAC murmur is often a puzzling factor to the beginners as well as examination-going students. It is sometimes so complex, that one gets different opinion from different physicians about a particular murmur, and less frequently about its etiology. A real murmur or bruit is an abnormal endocardial sound due to disease either of or close to the valve where it occurs, when it is often known as organic; some alterations in the state of the blood which by affecting its viscosity on the one hand and nutrition of the tissues of the heart and vessels on the other, produce conditions necessary for the development of a murmur, which is then known as functional.

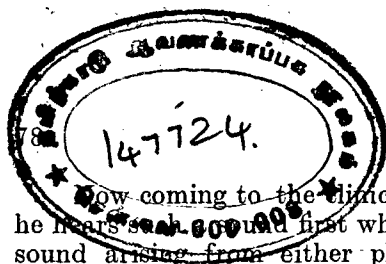
Why a murmur is produced and what factors take part in production of this sound are by no means clear. The possible factors concerned in its production are:—

- (1) The condition of the valves and the passage of the stream from a narrower into a wider channel—which may be relative or real.
- (2) Viscosity of the blood.
- (3) Velocity of the blood-stream.
- (4) Perhaps few more unknown factors.

The viscosity and velocity factors are very vague and we do not get murmur in all cases where there are extreme changes in either velocity or viscosity. All cases of severe anaemia do not show murmurs nor all cases of polycythemia. Same is true with a very fast heart rate, where a murmur is not always found. I have seen cases of anaemia with about 50% Hb and 2.5 Mil. R.B.C. per cmm. exhibiting a functional murmur while cases with much lower HB% and R.B.C. count, have not exhibited always a haemic murmur. This shows that there must be some additional factors over and above the ones quoted above.

\* Specially contributed to THE ANTISEPTIC.

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Now coming to the clinical aspects of murmur, one has to decide when he hears such sound first whether it is really an endocardial murmur or a sound arising from either pleura, lungs or pericardium. We shall first discuss the cardiac murmurs.

**Endocardial murmurs.**—These murmurs always have a definite relation to the events occurring in the course of the cardiac cycle and their time of production, character etc., varying with their sites of origin. These endocardial murmurs, are either organic or functional. In the so called organic murmurs where the valves or their surroundings are involved, a murmur is produced due to either obstruction to the onward flow of blood or from the leakage backwards through a closed but incompetent valve. The former is known as stenotic or obstructive murmur, and latter as incompetency or regurgitation murmur.

In functional murmur, there is no organic damage to either valves or the endocardium but murmur is produced as a result of either dilatation of a chamber with a relative incompetency of a valve or some changes in circulating fluid. Sometimes vibrations, which are produced in the walls of any cavity or chamber, due to sudden pressure changes in it, may be responsible in the production of such a sound. Corrigan's murmur in aortic regurgitation is due to such a factor.

When one auscultates a murmur the following details are to be taken into account:—

- (1) What is the time of its occurrence? Time relation with the systole and diastole of cardiac cycle is considered in relation with ventricles.
- (2) Where is it best heard? Site of maximum intensity.
- (3) Is it propagated beyond precordium and if so upto where?
- (4) What is its character? Rough—harsh—soft—blowing—musical machinery type, etc., etc.
- (5) Is it replacing any cardiac sound?
- (6) Is it altered in character etc., with change of posture or after some exertion?

I do not wish to discuss all these in detail as these things are well known to all, but I wish to emphasise a few points. The murmurs are best timed with the apex beat and next best with carotid pulsation. A loud murmur is often heard much away from its site of maximum intensity, and here one finds often difficulty in deciding whether this can be said as a true propagation or only as audible. This point is very difficult to decide in some cases, as in these cases murmurs are audible all over the chest and one should take all other factors into consideration before labelling it as its true propagation. Mere audibility should not be mistaken as propagation and vice-versa; and one should remember that any murmur loud in intensity may be audible in any place, and so it should not be considered necessarily as propagation. If this is borne in mind, mistakes will be averted in diagnosis of the nature of a murmur, where one relies more on its path of propagation. Remember that "audibility is not always its propagation in a murmur." I have often seen a loud presystolic murmur of mitral stenosis, without any evidence of incompetency, audible in axillary space. The maximum loudness of a murmur which has been produced at a particular valve usually occurs at a point where the valve sound would be best heard in health, but there are some exceptions to this rule.

Characters of a murmur depend upon the organic condition of the valve. Stenotic murmurs are harsh while regurgitation and functional murmurs are soft. The loudness of a murmur has no relation to the amount of damage to the valve, where it is produced. A very loud murmur is often far less serious than one so soft as to be just audible.

These few facts show that in a majority of cases, the murmurs exhibit their qualities mentioned in usual text-books but in some cases one comes across the abnormalities. As an example, an aortic regurgitation murmur, which is diastolic, is usually heard best at aortic area, but in some cases this murmur is best heard at mitral area and may not be even audible at aortic area and all other signs of aortic regurgitation are present. I had recently such cases in our wards. Before considering the exocardial and other murmurs, I shall describe in brief characters of a functional-vascular, or hæmic murmur. The exact mechanism of its production is by no means clear.

The chief characters are:—(1) It is usually systolic in time; (2) soft in character; (3) best heard usually at 2nd left intercostal space next best at mitral space; (4) does not replace any heart sound usually; (5) localised and not propagated outside; (6) may be modified on change of posture or exercise etc.

These functional murmurs are commonly met with in anæmia, chlorosis etc. In anæmia it is thought to be due to slight dilatation of pulmonary artery, beyond the valve ring and to a less viscous condition of the blood, which increases the vibrations set up by such a dilatation. The other possible factors in its production are:—

- (1) Fatigue or atonicity of valvular ring and papillary muscles.
- (2) Increase in velocity of blood.
- (3) Dilatation of valvular orifices.
- (4) Changes in blood—composition and viscosity i.e., polycythæmia etc.

**II. Other murmurs.**—This group includes the following murmurs:—

- (1). Pericardial murmurs. (2) Pleural. (3) Cardio-pulmonary murmurs.

1. *Pericardial murmur.*—This is produced as a result of friction between the two-layers of pericardium. The chief characters are:—

- (1) It does not correspond definitely with the events of cardiac cycle.
- (2) Generally it is more distinct in systole than in diastole but may be to and fro in character.
- (3) Sometimes the sound occupies the latter part of systole and early part of diastole without any pause between first and second elements.
- (4) Sometimes it is audible throughout the cardiac cycle.
- (5) It has no definite place of maximum intensity nor any selective propagation but usually loudest at 3rd left intercostal space.
- (6) It may vary from day to day.
- (7) May be associated with a distant thrill.
- (8) Intensity may be modified by the change of posture and on pressure.
- (9) If auricular area is involved, the to and fro rub may be replaced by a triple friction sound.
- (10) It is usually of superficial nature when heard over an area uncovered by lung.

(11) If pericardial sac contains air and fluid, a churning or water wheel sound may be heard.

(12) It is not affected by changes in breathing or holding the breath.

(13) The murmur is often accompanied by pain in chest and tenderness on pressure.

2. *Pleural murmurs*.—These are true friction sounds heard only when the patient breathes, modified with pressure and are usually associated with catching pain at that site, and have no relation with cardiac cycle.

3. *Cardio pulmonary murmurs*.—They are :—(1) Pleuro pericardial. (2) Pulmo pericaradial.

The sound produced due to diseased conditions in the neighbourhood of the heart may closely resemble the endocardial murmurs. These sounds are produced due to induction of abrupt movements of air contained in lung tissues and in bronchi as a result of displacement of the heart, or pressure upon the heart, or communication of heart's movements to the lung in an abnormal way through the adhesions. It is also possible that a slight degree of pleural friction may become audible during cardiac systole, if the roughened surfaces are situated near the special region of the heart.

Chief characters of these murmurs are :—

(1) They occur at the time of cardiac systole, beginning about the middle or near the end of it.

(2) Short in duration and are best heard during inspiration.

(3) Common site is just outside the apex beat—in some at the base of the heart.

(4) May disappear with postural changes.

The above discussion will be useful to diagnose the origin of a particular murmur, whether it is endocardial, pericardial or cardio-pulmonary etc. When the murmur is diagnosed as of endocardial origin then one has to find out its exact site of production i.e., from which valve it arises and lastly one has to find out its etiology. Most of the murmurs are very clear and easy to diagnose, but sometimes they are too complex to be correctly diagnosed by a casual observer. A patient may come with a single murmur, which is well heard at only one space, while the other patient may come with one loud murmur which might be heard equally well at all spaces, or a patient may come with multiple murmurs well heard at multiple places. Great difficulty arises in such cases for accurate diagnosis of each and every murmur, about its place of origin and etiology. Audibility of a murmur is not necessarily its propagation, and a loud murmur may be audible at some place, which is not its site of propagation. So the exact idea about the characters of all murmurs produced by various valve lesions is essential for correct diagnosis. I do not intend to discuss the characters of the different murmurs produced by lesions of different valves, as they are well known to all, but I shall now discuss the clinical aspects of various murmurs heard at various places and how to arrive at definite conclusion about their places of origin and etiology so as to avoid confusion. I shall briefly describe the salient features of various murmurs heard at various places.

I. *Murmurs at mitral area*.—The common murmurs usually met with at mitral area are :—(1) Presystolic murmurs; (2) Systolic murmurs; (3) Diastolic murmurs; (4) Mixed or double murmurs.

1. *Presystolic murmur at mitral* may be heard in (a) Mitral stenosis; (b) Tricuspid stenosis; (c) Austin-flint murmur; (d) Splitted heart sound mistaken for presystolic murmur.

(a) Murmur of mitral stenosis alone is easy to diagnose. It is a rough presystolic localised crescendo murmur, which is best heard at mitral area.

(b) Tricuspid stenosis is rare and murmur of this origin is often well audible at mitral area. Along with the murmur, presence of right auricular enlargement, enlargement of the liver, and other signs of tricuspid stenosis help us in accurate diagnosis.

(c) *Austin-flint murmur*.—This murmur is heard at mitral area, presystolic in time, often heard in full diastole, low pitch, rumbling and often with a thrill. It is less audible than mitral stenosis murmur. The murmur is associated with large heart and signs of aortic regurgitation. Exact mechanism is not clear but it is probably due to dilatation of cavity of left ventricle and so relative mitral ring narrowing, producing this murmur perhaps due to vibrations of mitral cusp during diastole when blood regurgitates in left ventricles due to incompetency of aortic valve.

(d) *Splitted first heart sound mistaken as presystolic murmur*.—Splitting of first heart sound is of great importance on account of its common occurrence and the frequency with which it is mistaken for the presystolic murmur of mitral stenosis. Indeed this is one of the most common errors which fastens unwarranted cardiac invalidism, from the misinterpretation of any abnormal auscultatory sign. Differentiation in such case can be done by its wider distribution than murmur and its greater intensity in the upright posture. The importance of heart sound in the clinical interpretation of various changes observed on auscultation has been more or less neglected compared to the importance given to the various murmurs which have attracted most attention. Careful attention to the qualitative changes and other abnormalities in heart sounds, such as splitting of sounds, addition of a fourth heart sound, and addition of a sound in late systole, is very essential for a correct clinical diagnosis and to avoid a gross error in labelling such a sound as a murmur, which means more cardiac invalidism for the patient. Significance of these additional heart sounds, triple rhythm, etc., were still ill-understood and I do not wish to go in detail about the mechanism of their production and conditions where they are produced, but I want to stress that one should avoid mistaking an abnormal or additional heart sound as a murmur. Other clinical data and findings will help in this matter.

2. *A systolic murmur at mitral area* is met with in—

(1) Mitral regurgitation.

(2) Anæmic conditions.

(3) Systolic murmur of tricuspid regurgitation is often well heard at mitral area.

(4) Murmurs in pericarditis.

(5) Systolic murmurs in congenital heart lesions, (specially patent ductus arteriosus, interventricular septal defects and interauricular septal defects) are often well heard at mitral area.

(6) Sometimes a systolic murmur is heard at mitral area in dilatation of heart, in acute fevers and other diseases.

(7) Often a soft variable systolic murmur is heard at mitral area in rapid heart, in effort syndrome, after severe exercise, and in hypertension

(8) Rarely a systolic murmur due to either aortic stenosis or pulmonary stenosis may be well heard at mitral area.

(9) A cardio-pulmonary murmur at mitral area may be missed for other systolic murmurs.

(10) Systolic murmur at mitral area is not always pathological. Innocent systolic murmurs at mitral area are reported even in quite healthy heart. The cause of this murmur is not clear.

The above list clearly tells us that there are many conditions in which we may hear a systolic murmur at mitral area. So when we get a systolic murmur at mitral area, we will have to find out the exact cause of its production.

(1) The systolic murmur due to pure mitral regurgitation is very easy to label. It is a soft, blowing descendo murmur, which is best heard at mitral space and is conducted towards axilla and even to back, and may replace first sound. It is often accompanied by systolic thrill. Other signs of mitral regurgitation are present.

(2) *Hæmic murmurs*:—Though usually best heard at pulmonary space, they are often well heard at mitral space. The characters of these murmurs are already discussed above.

(3) Systolic murmur of tricuspid regurgitation is better heard usually at tricuspid space and is accompanied by other features of this condition.

(4) Systolic murmur of pericarditis is also accompanied by a diastolic murmur, and this continuous type of murmur is heard all over the precordial area, does not replace any sound and is very superficial in nature.

(5) Systolic murmurs due to congenital heart lesions are better heard at second or third left intercostal space. These murmurs are often well heard at mitral area. History of congenital origin and other features are useful in diagnosis. In other conditions, the features of each condition help us in accurate diagnosis.

(6) *Innocent systolic murmurs at mitral area*:—With more comprehensive study, more precise interpretation of the mitral systolic murmur has been possible, and innocent or functional varieties of such murmurs are observed. About four types of innocent systolic mitral murmurs can be readily identified by clinical examination. They are:—

(a) Murmur of reclining posture:—Short-slow in character and best heard in reclining posture. Similar murmur may be heard at pulmonary area also. This type of murmur was observed by me in many healthy students at the University medical examination, during this year at Bombay.

(b) Murmur of upright posture:—same type as previous, but heard best when patient is in upright posture.

Both these murmurs are found in young persons and never after the age of about forty years.

(c) Third variety of this group is the innocent murmur in late systole. It is not uncommon and since it is always loud and long it is usually regarded as standing for organic heart disease. The murmur is late in systole, nearest to second sound than it is to the first.

(d) A fourth kind of innocent murmur, parasternal murmur. This is loud and long and best heard in the fourth intercostal space at the

left lateral border of the sternum. It is differentiated from the murmur due to ventricular septal defect by absence of thrill, or cardiac enlargement and normal E.C.G.

These two murmurs, unlike the first two, are found at any age and so are a common source of error in diagnosis. The mechanism and significance of these murmurs are not fully understood. Thus it is essential to be acquainted with the clinical characteristics of these murmurs and thus save the patients, who exhibit them from a life of unwarranted invalidism.

Thus the mitral systolic murmur arises from mitral regurgitation, aortic stenosis, aortic incompetency, hypertension, cardiac hypertrophy and also from functional and hæmic factors. The murmur in all these conditions has no individual distinguishing marks on auscultation, but the diagnosis is based on finding of other clinical signs, such as other typical murmurs of the respective conditions, pulse, blood pressure, cardioscopy, cardio-graphy etc.

3. *Diastolic murmurs at mitral area*:—They are:

(a) Murmurs of aortic regurgitation and pulmonary regurgitation sometimes best heard at mitral area. The other features of the diseases differentiate them from others.

(b) Presystolic murmur of mitral stenosis is sometimes audible throughout diastole.

(c) *Graham-Steel murmur*:—This murmur is heard in some cases of mitral stenosis, where due to back pressure in the pulmonary artery, the pulmonary valvular ring is dilated and a relative incompetency is produced which then causes a regurgitation murmur. Here the murmur is to be differentiated from that of aortic regurgitation murmur. Presence of mitral stenosis and other signs help in diagnosis.

4. *Mixed or double murmurs at mitral*:—Before going ahead, I tender my apology for repetition of some of the murmurs, already described. This is inevitable as some murmurs are often audible at various places and so are included in the discussion under various heads. As far as double murmurs are concerned at mitral area, one comes across either systolic and diastolic murmurs, or presystolic and systolic murmurs, or such other combinations. This might occur due to either production of various murmurs from one origin or murmurs from different origins. It is often very difficult to decide correctly which murmur arises from which origin and under all such circumstances one should take all other features into consideration before giving out final opinion. It is not my intention to make it more complicated here by discussing all possible permutations and combinations arising out of such origins, but I shall just discuss the important ones. The important combinations which we meet with at mitral space are as follows:—

(a) Presystolic and systolic murmurs.

(b) Presystolic and early diastolic murmurs.

(c) Systolic and early diastolic murmurs, etc.

(a) *Presystolic and systolic murmurs*:—These murmurs at mitral are met with in—

(i) Mitral stenosis with regurgitation. The other feature of this condition and characters of the murmurs are so clear that diagnosis here is easy.

(b) *Presystolic and early diastolic murmurs*:—This combination is met with in—

(i) A case where there is mitral stenosis as well as aortic insufficiency—both may be due to rheumatic origin or bacterial endocarditis etc.

(ii) In aortic regurgitation with left ventricular dilatation, sometimes a presystolic murmur is heard at mitral because of relative narrowing of the valve. So here we meet with an early diastolic murmur due to aortic regurgitation and a presystolic murmur due to relative narrowing of mitral ring. This presystolic murmur here is known as Austin-Flint murmur, mechanism of which is already discussed.

(iii) This combination is also met with when there is mitral stenosis with pulmonary incompetency, due to dilatation of pulmonary artery.

The early diastolic murmur due to pulmonary regurgitation here is known as Graham-Steel's murmur.

This shows that these murmurs have their different origins. In (a) Both the valves are affected together by common etiology; in (b) Only aortic valve is primarily affected by organic lesion; while in (c) Mitral valve alone is primarily affected.

The question at times is not so easy as we might think and often one is puzzled in deciding definitely the origin of such murmurs. The real trouble arises when a patient comes with an early diastolic murmur in a case of mitral stenosis. The following table will be useful for diagnosis of the exact origin of the diastolic murmur, due to early aortic or pulmonary valve lesion in such mitral stenosis cases:—

TABLE I.

| Signs                                  | Mitral stenosis with aortic insufficiency                                                     | Mitral stenosis with pulmonary insufficiency                                                  |
|----------------------------------------|-----------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------|
| 1. Stenosis.                           | 1. Moderate or absent.                                                                        | 1. More intense.                                                                              |
| 2. Pulse.                              | 2. Water hammer type.                                                                         | 2. Normal.                                                                                    |
| 3. Blood-pressure.                     | 3. High-systolic, low diastolic.                                                              | 3. Within normal limits.                                                                      |
| 4. Pulse pressure.                     | 4. Increased.                                                                                 | 4. Small or normal.                                                                           |
| 5. Systolic murmur in some cases only. | 5. Present over second right interspace.                                                      | 5. Present over second left interspace.                                                       |
| 6. Presystolic murmur.                 | 6. Best heard at mitral area and with all features of true stenosis murmur.                   | 6. Best heard at mitral area and with all features of true stenosis murmur.                   |
| 7. Diastolic murmur.                   | 7. Best heard at aortic space radiated along left sternal border. Often well heard at mitral. | 7. Best heard at pulmonary area—restricted to small area only. This is Graham Steel's murmur. |
| 8. On screening.                       | 8. Pulmonary artery slightly dilated or normal. Aorta slightly enlarged.                      | 8. Marked dilatation of pulmonary artery. Aorta normal or small.                              |
| 9. Pulsations on screening.            | 9. Increased pulsation of aortic arch.                                                        | 9. More marked with pulmonary artery.                                                         |
| 10. Hilar dance.                       | 10. Absent.                                                                                   | 10. Present in many.                                                                          |
| 11. Electro-cardiograph.               | 11. No axis deviation or slight right axis deviation.                                         | 11. Marked right axis deviation.                                                              |

(c) *Systolic and early diastolic murmurs*:—This combination is met with in—

(i) Mitral regurgitation with aortic regurgitation, mostly due to rheumatic infection, involving both the valves.

(ii) In cases of well advanced aortic regurgitation, where there is also secondary mitral incompetency, because of huge dilatation of left ventricle.

(iii) These murmurs may also be heard in cases of patent ductus arteriosus—usually they are best heard at pulmonary space.

Now we shall discuss the murmurs met with at aortic area.

II. *Murmurs at aortic area*.—Common murmurs, heard well at aortic area, are as follows:—(1) Systolic murmurs; (2) Diastolic murmurs; (3) Double murmurs.

These murmurs arise from either aortic valve or adjacent structures, such as ascending aorta, etc. The murmurs arising from the pulmonary valves are often well heard at aortic area, but other clinical features will help us to differentiate them, as far as their origins are concerned:

1. *Systolic murmur at aortic area*:—This murmur is heard in: (a) aortic stenosis of any etiology; (b) dilatation of ascending aorta; (c) aneurism of ascending aorta; (d) atheroma of base of the aorta; (e) Corrigan's murmur in aortic regurgitation; (f) in acute rheumatic infection, sometimes systolic murmur is heard at aortic area; (g) in many cases of hypertension; (h) hæmic murmurs are often well heard at aortic area.

I am not including in this list those murmurs which arise from other valves and are also well heard at this area, as other accompanying features will help in the diagnosis.

*Murmur of aortic stenosis* is rough in character, best heard at aortic area, systolic in time, and is conducted towards carotid arteries in the neck.

The murmurs in other conditions are associated with other features of respective diseases, and so they are easy to diagnose.

2. *Diastolic murmurs at aortic area*:—They are met with in: (a) aortic valve incompetency of any etiology; (b) dilatation of aortic ring in aneurysm of ascending aorta, thus causing functional incompetency of the valves.

Sometimes the diastolic murmur due to lesions of pulmonary valve, patent ductus arteriosus, Graham Steel's murmur, etc., is well heard at aortic space and so careful examination is necessary in such cases to detect the exact origin of the murmur.

3. *Combined murmurs at aortic area*:—Commonest combination is of systolic and diastolic murmurs. These murmurs are often known as "sea-saw" or "to and fro" murmurs. The common causes are:—

(1) Aortic stenosis as well as aortic regurgitation due to valvular lesion.

(2) Aneurysm of ascending aorta with aortic regurgitation due to dilatation of aortic ring.

(3) Aortic regurgitation murmur accompanied by Corrigan's murmur.

Combined murmurs due to other valvular lesions are also well heard here but are not included in this list. A continuous murmur of pericarditis is also well heard here, and may be mistaken for something else. Incompetency of aortic valve can occur without stenosis but one rarely meets with stenosis of aortic valve without incompetency. Characters of these murmurs, their maximum intensity, site, conduction, along with pulse characters, blood pressure and other cardiac findings will help us to arrive at a definite conclusion.

III. **Murmurs at pulmonary area.**—(Including those at III left intercostal space).

Most of these murmurs at these areas are either functional or of congenital lesions. A murmur at this space may be:—(1) Systolic; (2) Diastolic; (3) Combined murmurs.

1. *Systolic murmur at pulmonary area*:—Is heard in: (a) Congenital pulmonary stenosis; (b) Fallot's tetralogy; (c) Murmur due to patent foramen ovale and other inter-auricular septal defects; (d) Patent ductus arteriosus; (e) Inter-ventricular septal defects; (f) Pulmonary arteriosclerosis; (g) Functional murmurs in: (i) Anæmia; (ii) Dilatation of heart; (iii) Effort syndrome.

Murmurs of congenital lesions are best heard at III left interspace.

2. *Diastolic murmurs at pulmonary area*:—They are due to: (a) Pulmonary incompetency of functional origin or organic which may be: (i) congenital, or (ii) acquired; (b) Ayerza's disease (relative dilatation of pulmonary artery at the ring); (c) Graham-Steel's murmur; (d) Patent ductus arteriosus; (e) Pulmonary incompetency in some cases of inter-auricular septal defects.

3. *Combined murmurs at pulmonary area*:—The common causes of these murmurs are:—

(a) Patent ductus arteriosus.

(b) Patent foramen ovale and outer interauricular septal defects, where there is also pulmonary incompetency.

(c) In some cases of Ayerza's disease. Pulmonary arteriosclerosis.

(d) Arterio-venous aneurysm. (When an aortic aneurysm ruptures into pulmonary artery or vein or into superior vena cava). This murmur may be equally well heard at aortic area also.

The murmur of patent ductus arteriosus is known as machinery murmur as it is loud and continuous in character. This condition also presents water hammer type of pulse, large pulse pressure, low diastolic pressure and prominent pulmonary conus which is seen pulsating on screening.

The murmur of interauricular septal defect is usually in early systole and is best heard at IVth left inter space. Pulse is of small volume and B.P. is low. Diastolic murmur due to dilatation of pulmonary ring as a sequel to huge dilatation of pulmonary artery is often heard. On screening, huge dancing shadow of pulmonary conus is visualised. In Ayerza's disease, the murmur is soft and on screening the lungs will show typical mottlings. There are also signs of right-sided hypertrophy of the heart and some cyanosis, etc.

The characters of other murmurs are already discussed and so need not be repeated.

IV. **Murmurs at tricuspid area.**—These rare murmurs are either:—(1) Systolic, (2) Presystolic, or (3) Combined murmurs.

Systolic murmur is due to tricuspid regurgitation due to any cause. This murmur is soft and blowing in character, best heard at tricuspid area, often conducted upto liver area, may be associated with pulsating liver and is descendo in character. Presystolic murmur is due to tricuspid stenosis. Combined presystolic and systolic murmurs are due to both stenosis and

incompetency of tricuspid valve. Usually tricuspid valve lesions are rarely met with.

In the above discussion, I have tried to give all the clinical aspects of the common murmurs met with at various areas on auscultation, and their chief characteristics. I have tried to avoid repetitions and over lapping but it has been inevitable in some cases as many murmurs are often very well heard at other areas. It is always advisable to auscultate the heart keeping the patient in lying as well as sitting positions. Sometimes it is necessary to auscultate the heart after some exercise, before one gives his final verdict about a particular murmur. One should restrain in giving out final opinion about a particular murmur only on findings from auscultation as one should take other clinical findings such as pulse, blood-pressure etc., into account in exactly determining the origin of the murmur. Aortic murmurs are often very atypical and these precautions are very essential in such cases.

**Clinical investigation for its etiology.**—When we hear any murmur at any space, two questions arise: first about its exact site of origin in relation to heart, and second, about its pathological cause or etiology. We have already discussed how to diagnose the exact origin of a particular murmur, and now we shall deal with the second question. All these murmurs arising from endocardial causes, are either functional, or organic. The characters and general features of such functional murmurs are already discussed in former pages. The organic murmurs of endocardial origin are either of congenital or acquired origin. Congenital lesions are mostly on the right side of the heart. The murmurs might have been detected in early childhood and some conditions may be accompanied by cyanosis and clubbing of fingers, but not all. So history of the disease in detail, its duration, age of the patient, history of rheumatic infection or syphilis in past, and the present findings put together lead one to a correct conclusion. Conditions responsible for production of organic murmurs due to involvement of various valves and adjacent structures can be grouped as follows. This classification of etiology of valvular lesions is applicable to all the valves of the heart.

1. *Congenital anomalies of the valves*—i.e., (a) Congenital absence of the cusp; (b) Congenital stenosis; (c) Congenital defects etc., etc.

2. *Traumatic rupture of cusps*:—Specially in aortic valve, due to sudden excessive strain.

3. *Inflammation*:—Inflammation due mostly to infections, which may be acute or chronic. Chief of them are:—(a) Acute rheumatic infection; (b) Chronic rheumatic infection; (c) Acute bacterial endocarditis; (d) Sub-acute bacterial endocarditis; (e) Syphilitic infection on the valves and aorta.

4. *Degeneration*:—(a) Atherosclerosis of vessels and valvular involvement causing nodular sclerosis. This usually happens in old age.

5. *New growths*:—Very rarely a new growth near a valve may be responsible for the production of some murmur. Relative dilatation and relative narrowing of valvular rings are responsible often in production of murmur.

This includes most of the etiological factors damaging the valves.

**Additional investigations for confirmation**:—When one has presumed a particular murmur due to a particular cause affecting the valve, he would

like in some cases of doubt to confirm his presumption. So far as the murmur is concerned, the recent advance with "Cardiophonogram" will be of much help for accurate study of various murmurs. Electronic stethoscopes, which amplify the intensity of various sounds, will also be of much help in our future progress; but we have to await eagerly their practical use in India.

As far as pathological confirmation is needed, (besides our usual clinical history and examination electro-cardiogram, X-ray picture, etc.). some of the following examinations may be necessary in a particular case: Most important ones are: (1) Blood examination for Wasserman or Kahn test; (2) Blood for erythrocyte sedimentation rate; and in some (3) Blood for culture, if acute or sub-acute bacterial endocarditis is suspected.

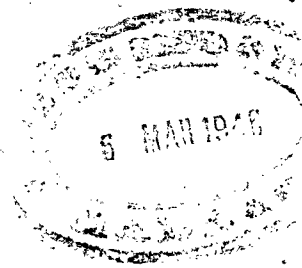
In conclusion, I wish one should remember that:—

- (1) All the murmurs are not of organic origin.
- (2) Presence of a murmur does not necessarily mean that heart is diseased, as often one comes across innocent murmurs.
- (3) Place of maximum intensity or audibility is not always the place of origin of a murmur.
- (4) Audibility of a murmur is not necessarily its conduction.
- (5) A valve can still be a diseased one in spite of the absence of an expected typical murmur at that site. Similarly a murmur can exist at any site even in the absence of lesion of that valve.
- (6) Severity of disease cannot be judged from the intensity and other characteristics of a murmur.
- (7) Murmur should not be taken as a splitted heart sound and *vice versa*.
- (8) One should not get satisfied with auscultation of heart in lying position only, but should do the same with patient in various different positions and even in some cases after some exercise if indicated and necessary.
- (9) One should not judge the origin and lesion from the murmurs alone, but should take all other clinical findings, such as pulse, B.P. screening, E.C.G. and other investigations wherever necessary into consideration before giving a final verdict.

I hope this much will be enough to have correct clinical approach in a cardiac case presenting any murmur, as far as the diagnosis of origin of the murmur and etiology of the particular valvular lesion are concerned.

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- |                                                  |                                          |
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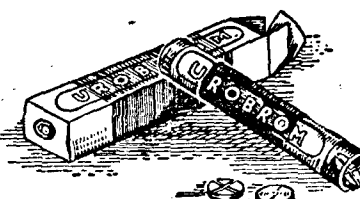
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## Prevention of Child-birth Maternal Injuries\*

RAO SAHIB S. M. TRASI, M.B., C.M.

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FOR any success to be achieved in the prevention of these injuries, a thorough appreciation of their causation is essential. For it is only by avoiding or eliminating their causes can they be prevented to any extent in practice. In a discussion on their prevention, their ætiology therefore claims our attention first. In general it may be said that they occur from lack of efficient aid or from mismanagement in labour, normal and abnormal. In this country of poverty-stricken villages—about 700,000 or more—in most of which there is no medical practitioner nor an efficient midwife, lack of suitable aid at child-birth is the main cause. The only help available in these villages is the barber *dai*, the 'traditional midwife', with her primitive methods, her only merit being her cheapness. Hence these injuries are very common in this country and their prevention is a difficult problem till efficient obstetric help is extended to these villages. The subsidised rural medical service recently started in some provinces is no doubt a belated experiment in this direction, but it does not touch the fringe of the problem, as it has as yet made little headway for various reasons which need not be gone into here. In the few cities and towns, where obstetric help of sorts is generally available, mismanagement is chiefly responsible for them. To realise how and to what extent it is so one must go more fully into their ætiology, the acts of commission and omission in the management of labour, normal and abnormal, that cause them, taking the injuries individually. The following maternal injuries may occur in child-birth: (1) Perineal; (2) Vaginal; (3) Cervical; (4) Uterine; (5) Anal and rectal; (6) Urinary; and (7) Pelvic floor injuries.

1. Of these the commonest are *perineal tears*. Its very position exposes the perineum to injury even in normal and more so in abnormal labour if care is not taken. Due to rigid bony boundaries (rami and symphysis) at the sides and front of the genital outlet, the full stretching force of the advancing head falls on the soft perineum behind, which bars its way, due to the abrupt terminal forward bend of the parturient canal. Its physiological softening in pregnancy—nature's protective provision—no doubt makes it more stretchable, but this has its limits and its safety depends on the birth of the head in full flexion with its smallest antero-posterior diameter (SOB =  $3\frac{1}{2}$ "") and circumference (11.8") through the outlet. Even so in its exit by extension its SOF diameter (4") and circumference (12.6") stretch the perineum over  $\frac{3}{4}$ " more (12.6"—11.8" = .8") and if this occurs during a pain when, to give better purchase to the contracting uterus, the pelvic floor muscles stiffen making the perineum tense it is very liable to tear:

(a) Crowning occurs often in "military attitude" and if suddenly born thus during a pain its OF diameter ( $4\frac{1}{2}$ "") and circumference (14.1") stretch the perineum  $1\frac{1}{2}$ " more still (14.1"—12.6" = 1.5") and severe tears even into the anus and rectum are liable to occur.

(b) If nature succeeds in flexing the 'crowned' head before its birth and saves the perineum, sudden birth of both shoulders together may over-stretch and tear it or extend minor tears.

\* Specially contributed to THE ANTISEPTIC.

(c) In OP and face births, OF and CV diameters, both  $4\frac{1}{2}$ " each have to pass through the outlet.

(d) In breech labours, with the urgent need in the interest of the child for quick delivery of the larger hard after-coming head through the cervix and outlet insufficiently dilated by the narrower soft forecoming breech, deep cervical and perineal tears are apt to occur; and if breech impaction, extension of head, arms and legs, etc., also occur the needed manipulations greatly increase the danger.

(e) When the forecoming head is stretching the outlet during pains, the vulvo-vaginal sphincter (levator ani + bulbo-cavernosus) offers strong resistance and if powerful pains drive the head or it is dragged, through at the time, the sphincter and perineum are both liable to tear. Such are the factors in perineal tears. Minor tears frequently occur in normal labour, few primiparæ escaping. But they are more often than not due to neglect of care of perineum by midwives, few of whom pay attention to it. Hence major tears too are not rare in their practice. But most major tears occur in abnormal labour, spontaneous and operative, and in the latter, from forcibly dragging the head prematurely through the outlet, or rough manipulations to bring down extended legs or arms.

2. *Vaginal tears*:—Lower vagina is always involved in perineal tears. Cervical and uterine tears may run into its top. Its middle may be torn from careless use or sudden slipping of instruments in craniotomy, forceps or decapitation operations or using the forceps to rotate or lever the head out. Forcing the hand in for version has torn the vagina across (CUTHBERT LOCKYER).

3. *Cervical injuries*:—Minor cervical tears occur in most primiparæ even in normal labour. As these cause no symptoms, the cervix is rarely inspected and they escape notice. Severe tears occur in precipitate labour, or in dragging the head or forcing hand or forceps through partly dilated cervix or its hurried manual or instrumental dilatation, especially in placenta prævia in which the cervix is friable. Such tears may extend up into uterus, down into, or laterally into broad ligament, or in front into bladder.

4. *Uterine tears*:—Uterus may rupture in unattended obstructed labour or careless operations, as version, forceps, cephalotripsy or decapitation, when it is in imminent or actual tonic contraction, or in forcing hand or forceps or dragging the head through a contraction ring or imperfectly dilated cervix. Rough attempts at manual removal of adherent placenta, especially placenta accreta, or vesicular mole, curetting in the latter and indiscriminate use of pituitrin are other causes. Old weak Cæsarian scars or an interposed or high ventrifixed uterus may burst in labour, while vigorous, hurried and premature attempts to express the placenta or extract it by forcibly pulling on the cord, when the uterus is relaxed or atonic, may cause inversion of uterus.

5. *Anal and rectal tears*:—These often occur in severe perineal tears. Only a few outer fibres of the anal sphincter may be torn leaving it weakened or it may be torn right through, the tear running into the rectum. A carelessly used perforator may slip and penetrate into the rectum.

6. *Urinary injuries*:—Careless use of instruments in craniotomy, decapitation or forceps, or using the latter to rotate or lever the head out may injure the bladder. Front uterine or cervical tears may extend into

it. The full bladder may burst if the head is forcibly dragged past it. But the usual cause of bladder injury is prolonged impaction of head in the pelvis in obstructed labour, in which adjoining bladder and vaginal or cervical walls get crushed between the head and symphysis and later slough away leaving a fistula. The urethra may, though less often, be also so injured. Occasionally the entire urethra with or without the trigonum may be destroyed. Both bladder and urethra may be injured in careless and difficult forceps, symphysiotomy or pubiotomy operations. Prolonged high obstructions and impaction of head may occasionally injure the ureter and cause a uretro-vaginal fistula. The bladder sphincter may be injured if the bladder is not quite empty when the head is born or extracted. The remnant urine with the bladder wall around it, is then forced down by the advancing head, and the urine under pressure from behind, forcibly stretches open this sphincter, thereby overstretching it and tearing some of its fibres in its efforts to escape out, leaving it permanently relaxed and weak, with resulting strain incontinence.

7. *Pelvic floor injuries*:—These are due to attempts to drag the head through a partly dilated cervix or a contraction ring down the uterus itself due to the resistance in front and severely strain and overstretch its supports and the pelvic floor tearing some of their fibres. Forcibly pressing down the uterus in vigorous premature attempts to express the placenta has the same effect. Hurriedly dragging the head through the outlet overstretching the pelvic floor muscles and fascia tearing some of their fibres and also the vulvovaginal sphincter, and thereby weakens the pelvic floor and leaves a gaping vulva leading sooner or later to genital prolapse, vaginal or uterine or both.

**Preventive measures.**—In the light of this ætiology prevention of these injuries may be discussed under: (A) Normal labour and (2) abnormal labour.

**A. NORMAL LABOUR:**—The two most important precautions in normal labour are: (1) Care of perineum; (2) careful management of third stage.

1. *Care of perineum in normal labour* is well described in text-books, but will bear repeating for its frequent or almost invariable neglect in practice. Its principle is, to deliver the head by its smallest diameter through the outlet between pains, when the perineum is relaxed.

The actual steps are: (1) After head-crowning, advance of sinciput is checked during the next pain or pains, by pressing on it with the left hand fingers just in front of the perineal edge, till the occiput which is left free to advance is born and the neck is behind the symphysis. This promotes full flexion and brings the S.O.B. diameter of the head into the outlet.

(2) Then, when a pain has just subsided and the perineum is relaxed, the head is gently pushed out with careful pressure of the base of the right hand or thumb and fingers on either side midway between coccyx and anus.

(3) If a pain comes on at this time the advance of the head is checked with the left hand or both hands interlocked if needed, the patient is asked not to strain but cry out, and as the pain subsides the head is delivered as above. These steps are best carried out with less risk of perineal tears, with the patient in the left lateral obstetric position, the attendant stooping over her from behind and passing his or her left hand from the patient's front, between her half flexed thighs. Direct hand support of the perineum is liable to reflexly excite pains and determine tears.

(4) After the head is delivered and the next pain has turned the front shoulder forward to the symphysis, the head is gently drawn to the mother's front to deliver the hind shoulder and then back to deliver the front one. Fundal pressure helps at this stage, one hand on the fundus the other managing the head. If hands appear before shoulders, they are gently drawn out. The trunk follows the shoulders without hindrance. This technique is simple and if midwives are well trained in it as an *essential part* of the management of normal labour, far fewer perineal tears, minor or major, will be heard of in their practice in this country. This raises a rather ticklish question. All authorities stress the great importance of primary repairs of perineal tears "as the raw surface acts as breeding ground for numerous organism and also acts as a focus for local and general puerperal sepsis." "Deep lacerations involving levatores ani weaken the pelvic floor" (Ten Teachers) and lead to genital prolapse. "Even tears less than one inch in depth are liable to cause sepsis and lead to relaxation of vaginal outlet and prolapse of vaginal walls" (CUTHBERT LOCKYER). "Any tear extending beyond the fourchette should be repaired immediately to prevent infection and formation of puerperal ulcer and prevent subsequent gynæcological ailments from loss of support to pelvic floor due to tearing of levatores fibres" (TWEEDY). The combined text book is even more emphatic than these: "In all instances the lacerations should be repaired, no matter how small they may be. It is very important to preserve the vaginal sphincter as it prevents contamination of infection spreading up." But how is this to be achieved in this country where, unlike as in the West, most normal labours and many abnormal spontaneous ones are attended by only midwives and therefore perineal tears, more frequent than in the West are left unrepaired as surgical help is rarely called in or available at the time? This state of affairs, a fruitful cause of immediate morbidity and later of various gynæcological troubles has to be remedied. The only solution of this problem in the present state of affairs in this country seems to be to train midwives themselves to put in the needed stitches in their cases, except in the 3rd degree tears which are beyond their capacity. This suggestion is not really so ridiculous as it may at first sight appear. Women are generally adepts in stitching and sewing. Midwives ought therefore to be able to learn the correct technique of repair of 1st and 2nd degree perineal tears if properly trained. Some may not do it quite well, but for that matter, neither can all medical attendants escape such criticism. The writer has seen instances of shabby stitching by medical attendants, skin of one side in apposition with mucous membrane of the other with two open raw surfaces—ulcers—one in the vagina and one in the perineum! If some midwives stitch not quite well the patient will in no way be worse than this or if unstitched. At any rate the experiment is well worth trying. Nurses and midwives are now permitted in many hospitals, to give hypodermic injections of potent drugs like Morphine, Hyoscine, Digitaline, Strychnine, Adrenaline, Ephedrine, Emetine etc., to patients though under instructions left by medical attendants in charge. This suggested innovation is only one step further but not so risky. For with injections one cannot guarantee that some nurses and midwives may not resort to them independently to the detriment of the patients.

2. *Care in the 3rd stage*:—This too needs stressing as premature attempts at placental expression are far too common in practice. It is a great temptation to impatient midwives and practitioners to hurry the 3rd

stage with premature attempts at Crede's expression or card traction to save time. This is inviting disaster. The patient's safety is infinitely more important than the attendant's time or convenience. HASTY CREDE's expression "squeezing the uterus like squeezing lemon" as TWEEDY puts it, is apt to set up irregular contractions, cause partial separation of placenta, loss of retro-placental hæmatoma, an important factor in natural placental separation, and so interfering with the normal process, cause retention of placenta or pieces, PPH, inversion of uterus and injury to uterine supports and pelvic floor. Natural separation of placenta takes 15 to 30 minutes, sometimes longer. "In a series of 2,000 cases in the Rotunda Hospital, placenta was retained in 93, but only 4 of these needed manual removal. Patients showing no bad symptoms have been kept in the labour ward up to three hours before placenta could be expressed. Such delay is justified if it saves manual removal with its risks of shock and sepsis" (TWEEDY). The only two conditions calling for prompt action in the 3rd stage are shock and hæmorrhage (PPH). In the absence of these, midwives should be taught to wait for and recognise the cardinal signs of natural placental separation.

- (1) Hypogastric bulging not disappearing on catheterising patient.
- (2) Uterus rising higher, feeling smaller, firmer and more movable.
- (3) Vulval-side cord ligature dropping lower (apparent lengthening of cord).

(4) Cord lengthening more on pressing on fundus and *not* being drawn in again on removing the hand from the fundus. This last is the most reliable sign. When these signs appear the uterus is first massaged through the abdominal wall into firm contraction, and placenta expressed by Crede's method. After natural separation the placenta falls into the lower segment pushing up the now fully retracted upper segment which rests on top of it (cause of signs 1 and 2) and little force is needed to expel the now free placenta, by using the firm upper segment, as a piston to push it out without any risk of injury to uterine supports or pelvic floor or of uterine inversion. Cord traction to extract the fully separated free placenta is less objectionable and may be combined with Crede's expression but is rarely needed.

**B. PRECAUTIONS IN ABNORMAL LABOUR**:—Timely detection of malpresentations and positions ante-natally or early in labour as per opportunity, and if not, in uncorrected and uncorrectable cases and in contracted pelvis, care in the choice and performance of needed operations, especially avoidance of excessive force, are the precautions needed. (a) Thus breech, OP, face, brow and transverse presentations are changed into more favourable ones. How and when this is best attempted for success is described in text-books and need not be repeated here. (b) In minor pelvic contraction with slight disproportion trial labour at full term, with forceps later in Walcher's position if needed. (c) In marked disproportion, premature labour at a suitable time as judged by Munro Kerr's method or Cæsarian at term, and if this is refused or not possible, embryotomy. In breech labours there are two occasions when special care is needed to avoid injuries. (a) To the cervix as the head is extracted through the brim, and (b) to the perineum as the head got through the outlet. This is due to the fact that breech traction usually adopted to deliver the trunk in breech labour, however carefully applied, tends to cause extension of head and arms and a larger diameter of the head; with often the thickness of arms added, has to be quickly got through the imperfectly dilated cervix

and out-let. This is best avoided by relying more on fundal pressure during pains which keeps the head and arms flexed, than on breech extraction alone—one hand on the fundus with the patient on her back, the other managing the breech, if no assistant is available. The trunk is thus delivered more by expression than extraction alone. When the trunk is born it is allowed to hang down for 2 or 3 minutes, (wrapped in warm dry cloth to prevent premature inspiration) as advised by BURNS. Its weight keeps head flexed and the delay allows time for it to mould and pass more easily through the brim. Walcher's position often helps at this stage and in difficult cases the (after coming) head is more easily and safely extracted with forceps as it maintains the head in flexion and avoids strain on the neck during extraction.

The danger to perineum is from hurriedly dragging the head through the outlet or forcible attempts to extract breech with extended legs impacted low down. Fairbairn's method of head extraction avoids hurry in its delivery. When the head reaches the outlet, and neck is behind the symphysis, the trunk is swept by the feet, towards the mother's front, till the foetus is upside down and its mouth and nose are free in the outlet. The head is now in the grip of the outlet. With the child thus upside down, one can wait a few minutes till the outlet relaxes and the perineum feels soft; the head is then easily rolled out by titling the trunk on to the mother's abdomen, with little risk to the perineum. As the mouth and nose are free in the outlet, open to the air, the foetus can breathe without danger of foreign matter getting into its air passages in its upside down position, as such matter escapes freely by the mouth and nose and is easily wiped off.

In breech impaction low down with extended legs, difficulty is due to the legs acting like splints, preventing latero-flexion of trunk needed for its birth through the outlet due to the forward direction of the latter. Double medio-lateral episiotomy as advised by Mc'DONALD allowing the perineum to fall back as a valve, and thus removing the chief obstruction overcomes this difficulty and makes extraction easier and saves the perineum from severe medical tears with their attendant danger to the anus and rectum. It is also useful in OP and face births in which also severe perineal tears are liable to occur.

**Precautions in operations.**—There are two precautions needed in all obstetric operations: (1) never to pass any instruments into the parturient canal without a guarding hand in it to guide the instrument and protect maternal soft parts; (2) never to do any operation without emptying the bladder just before. Of the operations the commonest and most responsible for maternal injuries is the forceps operation, as it is often needlessly done or even when contra-indicated, and often with brutal force. One also often sees the instrument used with little regard to its legitimate function. Text book description of its uses is perhaps partly responsible for this. Text books describe its 'sections' or 'uses' as: (a) tractor; (b) rotator; (c) lever; (d) compressor; (e) uterine stimulator (dynamic action), with a qualifying remark that its 'real' and 'most important' use is as tractor and without considerable experience the other uses are very risky to the patients. If so, why give them the dignity of its 'uses' or 'actions' and burden the instrument with such risky questionable uses? Why not mention traction only for which in fact it was designed? That some expert operators from long experience acquire exceptional skill with the instrument and may at times use it as a rotator or lever to extract the head in difficult

cases, is no valid reason to include such uses in its actions. The objection to their inclusion is its psychological effect on the minds of students, house-surgeons and young practitioners to regard them as legitimate uses and attempt them in their practice without the needed experience and skill, to the detriment of the patients. A passing reference to them and their technic in their relevant sections *e.g.*, as rotator, in OP and MP cases and as lever in Walcher's position in cases of minor disproportion just to get the head past the obstruction, with due warning against such risky uses by the inexperienced, may perhaps be less objectionable. In such rash attempts by the inexperienced, the instrument is very liable to slip suddenly and cause various tears in the mother. The vagina, vulva, perineum and even the cervix and uterus may be, and are often extensively, torn, the foetal head severely compressed, causing various injuries such as facial paralysis, cephal-hæmatoma, scalp tears, skull fractures, meningeal and cerebral tears, intra-cranial hæmorrhages, etc., from the stress. Compression is thus not a 'use' but definitely harmful except in craniotomy when it is sometimes helpful in the absence of compression instruments. Forceps is applied before or after perforation, compression during extraction expresses enough brain matter for easy delivery.

Uterine stimulation (dynamic action) is after all but a side effect, often observed also in internal version on passing the hand into the uterus, to the acute discomfort of the operator, who has to remain passive as long as it lasts, lest he rupture the uterus. It is certainly not among the uses of the hand in that operation, nor is it availed of even when it may be a rational indication in primary uterine inertia (sluggish uterus) in which forceps is most often used, not for its dynamic action, but as tractor. Even the words 'actions,' 'uses,' 'tractor,' are not a happy choice for the 'function' of the forceps. Actions and uses may be 'legitimate' or otherwise, while 'function' connotes only 'legitimate' use and is a better word to express the use of the forceps. 'Tractor' (traction = pulling, dragging) conveys a subconscious suggestion of force and the operator is apt to instinctively increase the 'pull' on meeting with resistance, too often ending in using undue force with often disastrous results. If due to long usage, the word has to be retained it is better to prefix an 'ex' to it as 'extractor' does not convey the same suggestion of force. Students and house-surgeons will have observed in the wards and out-patients' departments that in 'extracting' foreign bodies from the ear, nose and throat, skill and not force is used by the successful surgeon and is the secret of his success while operators who use force not only fail in their object but often severely injure the patients. Again to get a blind man out of a winding alley interspersed with obstructions, one does not drag him along by main force like a log of wood but carefully leads him along, grasping his arm and guiding or leading him past the obstructions, just so the obstetric forceps as its name 'forceps' implies, is meant to grasp the foetal head and guide or lead it out or as the combined text book aptly has it 'to assist it past any slight obstruction' in *minor* disproportion, and not to drag it through by main force. A certain amount of 'pull' has to be used no doubt, but it should be the absolute minimum needed, just the amount the uterus lacks to effect natural delivery. 'It must be no more than what a man of average strength can exert with his two fore-arms' (COMYNS BERKLEY). The writer would restrict it to *one* forearm or rather arm, for the muscles of the whole arm participate in the pull. The best guide to the safe limit of the force to be used is one's muscle sense in the pulling arm. The least sign of

fatigue or aching in the arm muscles from the strain means that the limit has been reached. There is no other practical measure of safe limit. Hence the advantage of using only one arm. It avoids the risk of consciously or unconsciously using undue force associated with the use of both arms, especially if the operator is a powerful individual. 'It is certainly quite unjustifiable for the operator to employ such force as can be obtained by counter pressure with his feet against the side of the bed' (COMYNS BERKLEY). This however is exactly what many operators do, even some teachers. It was the favourite practice of a certain professor, though often the head burst its way out suddenly causing 3rd degree perineal tears. Another teacher, while condemning this practice in a publication, strangely enough advised as the correct method, standing with feet apart and leaning back 'as in a tug of war' with handle in both hands! One fails to see any essential difference in these two methods except in the position of the operator. One sits on a seat and takes purchase with his feet against the bed, the other stands and takes purchase against the floor. Both pull with their two hands and both lean back bringing their full weight to bear on the instrument. 'The amount of traction which can be exerted by the forceps is enormous, but as a rule the operation is badly performed and the conditions are unsuitable when extreme force is employed' (combined text book). One can well imagine the effect of adding the operator's weight to this 'enormous traction.' Serious injuries—severe tears of the perineum, anus,

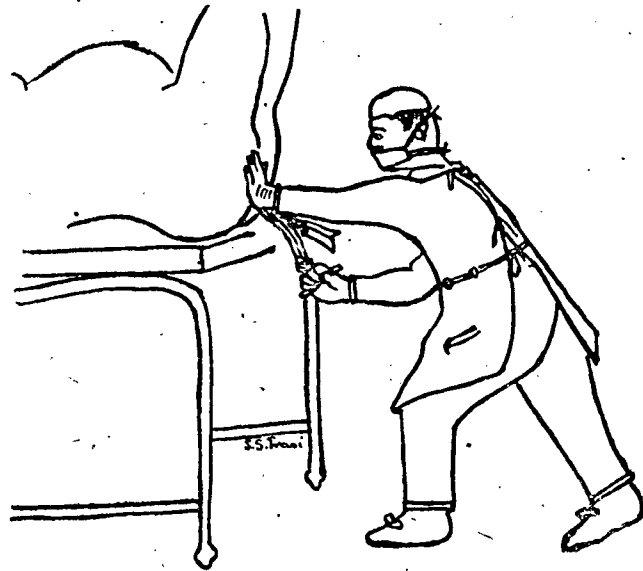
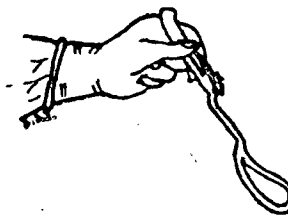


FIG. 1.

rectum, cervix, uterus and soft parts at the brim right down to the bone, etc., often occur. Many mothers die of shock, hæmorrhage or sepsis, and many children are lost. The forceps has suddenly slipped, the operator toppling back on to the floor with a thud, and a large quantity of blood splashed over and around him. All such dangers can be avoided if instead of leaning back, one leans forward during extraction (Fig. 1). This avoids the risk of adding one's weight to the pull. Counter pressure is applied with the left hand against the patient's left buttock (Fig. 1). When the head is brought down to the 'Crowned' position, the instrument is removed and the head delivered as in normal labour. But there is difference of opinion on this point. The ten teachers advise removal as safer for the perineum. COMYNS BERKLEY advises removal even earlier, when the head has been brought down on the perineum, and to allow the uterus to expel the child. But the combined text book advises its retention on the score of better control of head with it to

lessen pressure on the perineum. As even the small thickness of the two blades added to the head may turn the scale against the perineum especially in primiparæ with small outlets, their removal is advisable in such patients. In OP and face deliveries extractions should be slow and deliberate as the outlet is approached or the head may suddenly burst its way out, widely tearing the perineum. Carefully 'ironing' the perineum before applying the forceps often saves the perineum and if not enough, medio-lateral episiotomy on one or both sides in difficult cases saves it from severe medial tears with their attendant danger to the anus and rectum.

Again maternal injuries may occur not only during extraction but even during application of the forceps if care is not taken to avoid force. In the use of surgical instruments skill counts more than force. It makes all

FIG. 2.  
Pen-hold.

the difference between a neat safe operation and a clumsy crude dangerous one. Skill comes with experience, but avoidance of conscious or unconscious undue force in their use is possible for all, with correct handling of instruments even in minor operations. Thus in passing the silver catheter in the male—a minor operation—the catheter is held lightly by its handle-end between the thumb and the index and the middle fingers like a pen, the tip slipped into the meatus and by gently raising the handle-end the

instrument is allowed to glide smoothly into the bladder by its own weight along the curved male urethra. So also in the application of the obstetric forceps each blade is held lightly by its handle in a 'pen hold' (Fig. 2) and allowed to slip into position smoothly by its own weight, gently moving the handle in the needed direction. It is interesting to observe the parallelism in these two operations—one a minor and the other a major operation in 'different sexes: (1) The male urethra and the parturient canal are both curved soft passages partly without and partly within the pelvis. (2) The

silver catheter and the obstetric forceps, one a light narrow tube, the other a heavy two bladed instrument, differing in shape, size, weight, construction, design and function have both a pelvic curve. (3) Both should be held lightly in a 'penhold', to avoid force in passing them in.

(4) Both are allowed to slip in smoothly by their own weight.

**Technique of forceps application.**—As this is well described in text-books, only points that make for safety will be considered.

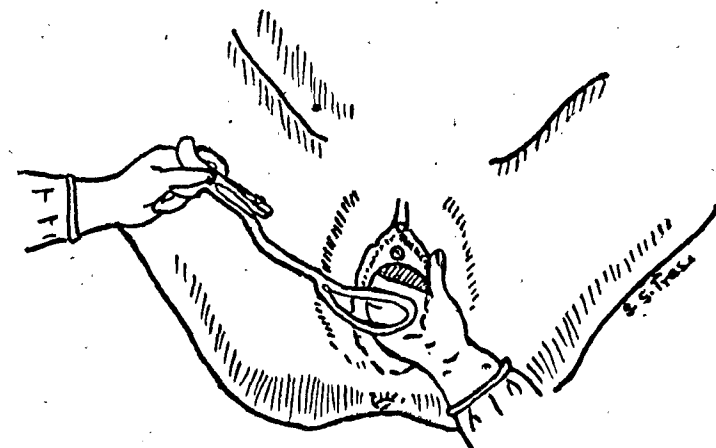


FIG. 3.

The forceps may be applied, with patient either in the left lateral or dorsal obstetric position, across or at the foot end of the bed. For patients on the floor as in many poor houses in this country, left lateral position only is possible. The blades may be passed from the sides of the pelvis, or

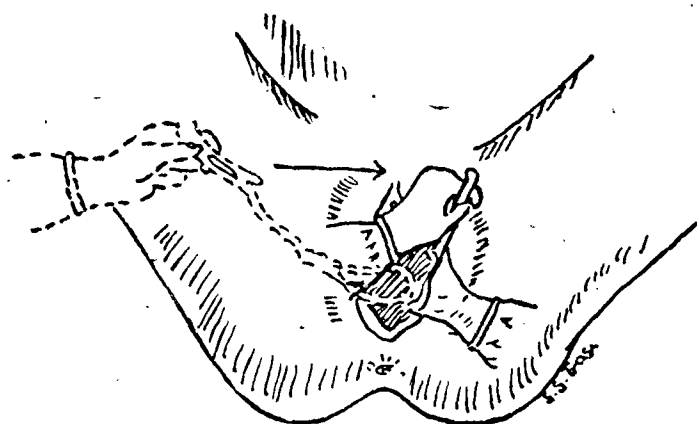


FIG. 4.

from the sacral hollow behind. To pass them in from the sides, each blade is held lightly in a penhold with the handle parallel to the opposite thigh, the tip of the blade resting on the guarding fingers in the vagina on the side of the blade to be passed (Fig. 3 vide page 97). The handle is then

gently moved straight to midway between the thighs (Fig. 4) and swept in a curve to the perineum across the corresponding thigh (Fig. 5). This method is generally used by beginners and those with limited experience,

perhaps because of the names of the blades, left and right—lower and upper. Passing the blades from the sacral hollow is more artistic and skillful and also easier. Each blade is held at right angles (perpendicular) to the symphysis, the tip of the blade resting on the guarding fingers in the sacral hollow (Fig. 6 vide page 99). The handle is gently moved straight back in the direction of the perineum and when half way back (midway between the thighs) (Fig. 7 vide page 99) swept in a curve to the perineum across the opposite thigh (Fig. 8 vide page 100). It



FIG. 5.

may be observed that the final movements of sweeping the handle in a curve across the thighs are diametrically opposite in the two methods. In the side application of the forceps, the handle is swept in a curve across the corresponding thigh and in the sacral application, across the opposite thigh. Thus used as a lever of the first order the blades slip smoothly into

position without the exercise of any force. The 'penhold' of the handles makes conscious or unconscious exercise of any force physically impossible.

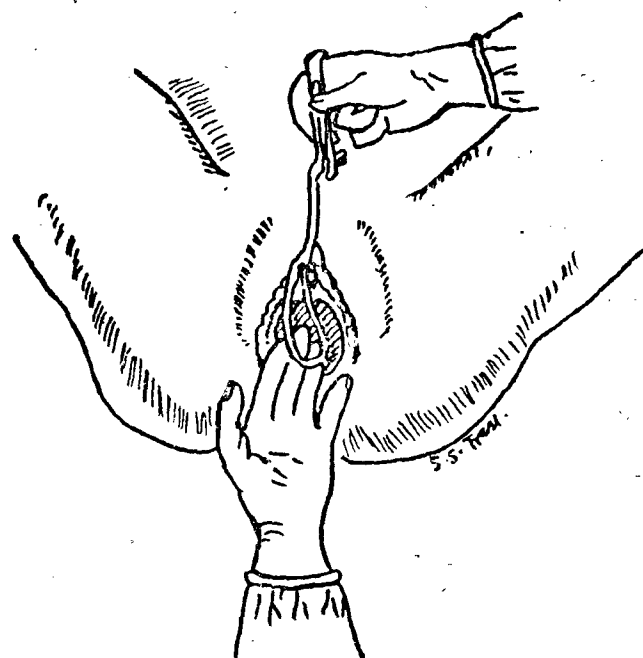


FIG. 6.

forced the forceps blades right into the abdomen through the vault of the vagina, as COMYNS BARKLEY mentions. If the forceps is forced thus through a partly dilated cervix, contraction ring or into a tonically contracted uterus both cervix and uterus, are liable to be torn. With the 'penhold' such disastrous accidents are physically impossible. Hence it is the only safe hold for passing in instruments through 'soft passages, especially, the heavy obstetric instruments. One point remains to be mentioned, and an important one. Young practitioners with limited experience should, as far as possible, avoid high forceps operations, as they are

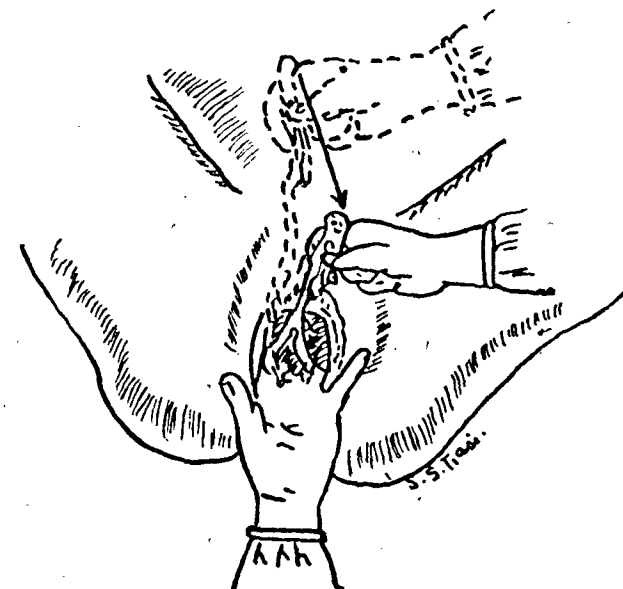


FIG. 7.

The sword grip (Fig. 9 vide page 100) depicted in certain books and the dagger grip used by certain operators are both risky, being full hand grips, and with them powerful force can be and is often consciously or unconsciously exerted by impatient operators on meeting with any resistance. Those who boasted that they never failed to pass the silver catheter in the male even in stricture and enlarged prostate cases have, with such grips forced it right into the rectum and careless operators have

generally called in, if at all, only too late, at least in this country, when the uterus is in imminent or in actual tonic contraction and on the verge

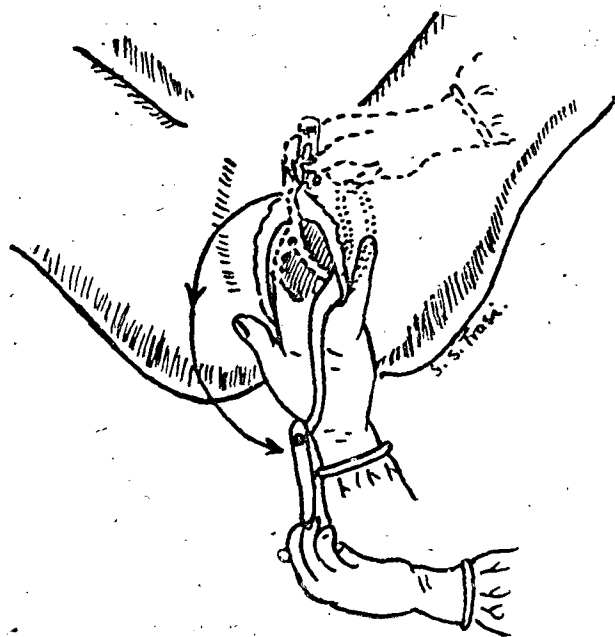


FIG. 8.

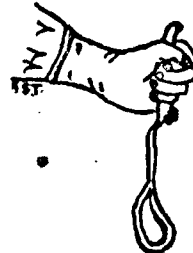
even if the child is alive. As the forceps operation is the commonest and most responsible for maternal injuries it has been discussed at some length.

The other operations, less common, and therefore less responsible for injuries in the mother, do not need such lengthy discussion.

**Internal version.**—This should never be attempted: (1) In impending or actual tonic contraction of the uterus; (2) If the head has passed through the brim; (3) In failed forceps cases. On the first two issues there is no difference of opinion, but on the third there seems to be. The

only, *raison d'être* for version after failed forceps is or should be in the interest of the child if it also can be saved. But it is rarely born alive in such cases and the mother will have been needlessly subjected to the risks of uterine rupture and sepsis. The writer was once present at a meeting where an enthusiastic young obstetrician read a paper advocating version after failed forceps, and presented a series of his own cases—under a dozen. In not one of them was the child delivered alive. That neither uterine rupture nor serious sepsis occurred in the series as he claimed, is no argument in its favour so long as its main object failed. Strangely enough he was supported by a famous authority present at the meeting, though

of rupture and the fetus dead or dying. In such cases attempting forceps is inviting rupture of uterus and even if the child is delivered alive, it will be born in white asphyxia and soon die. Thus both mother and child will be lost. The rule in obstetrics is, when the mother's and child's interest clash the mother's interests are paramount. If she lives she can have children again, but if she is sacrificed for the sake of the child, it cannot have another mother. Hence the correct procedure in such cases is embryotomy

FIG. 9.  
Sword grip.FIG. 10.  
Dagger grip.

he acknowledged the failure of the main object in the series. The writer once witnessed such an operation attempted in a case of marked disproportion. The child was born dead and the mother died from rupture of uterus and PPH. This danger is always there. For the operator rarely gives up forceps in time in the hope that a little more pull may succeed till he is tempted to use extreme force and persist too long and by the time he realises, only too late, that forceps is useless in the case and tries version the liquor Amnii will have all escaped and the uterus retracted and contracted tightly on the child, which is generally already seriously injured by the prolonged pressure of the forceps and extreme force used, and both mother and child are lost.

In cases in which there are no such contra-indications (1, 2, 3 above): (1) The patient should first have a Morphia injection and be fully anaesthetised to secure full relaxation of the uterus; (2) The uterus should be supported with a hand on the fundus pressing it down while the other hand is being passed in; (3) If a pain comes on as the hand is being passed or after it, no attempt should be made to force the hand higher or turn the child till the pain has subsided and the uterus is lax again; (4) After turning, if the cervix is not fully dilated, delivery should not be hurried by dragging the child through forcibly, but left to nature, and a small weight, a lb. or two, may be attached to a leg of the fetus, with a bandage and hung over the end of the bed; (5) In the delivery of the after-coming head, precautions mentioned under breech labours should be observed.

In *cephalotripsy*, *decapitation*, *cleidotomy*, *evisceration* and *spondylotomy*, little more need be said than due care in passing the instruments and during the operations, to avoid slipping of the instruments and injury to the mother's soft parts.

Pubiotomy, symphysiotomy, and Caesarian section are not suitable for general practice as they require special help, apart from the fact that they are best done only by experts and those with special training.

In precipitate labour the practitioner has generally little chance of being present since he is either sent for or arrives only 'after the event.' If he has such a chance it is generally advised that he should 'ask the patient not to strain'. This is but a 'counsel of perfection'. For the patient cannot help straining with her over-powerful pains. It is better to anaesthetise her; a Morphia injection too is of help to control the strength of the pains. The head should be checked in its rush at the outlet, with both hands interlocked and the head delivered slowly to allow enough time to the perineum to relax and yield without tearing.

The writer owes an apology for the long delay in the appearance of this article. It should have appeared soon after its first part 'Child-Birth Maternal Injuries' published in the April 1943 issue of the *Antiseptic*. But various unavoidable circumstances in the domestic sphere of the writer combined to make this impossible.

## Infantile Hepatic Cirrhosis

(Conditions described as infantile biliary cirrhosis; sub-acute toxic cirrhosis; and infantile liver.)

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(Continued from page 35 of January '46 issue).

**The clinical picture.**—Infantile hepatic cirrhosis contributes a good bit to infant mortality in India. For the city of Madras alone, the number of deaths due to this disease is 166 for 1938, and 186 for 1939. Deaths from cirrhosis of liver other than alcoholic in both adults and children amount to 244 in 1940 and 112 in 1941. These figures are not of much value as the death certificates are not always countersigned by the medical practitioner. It is therefore necessary that the clinical picture of infantile hepatic cirrhosis should be made as clear as possible so that there should be as little confusion as possible in the diagnosis.

The disease begins insidiously. A child is sometimes diagnosed as suffering from infantile hepatic cirrhosis in the course of a routine examination, as during the investigation of an irregular fever, or stubborn bronchitis or some form of digestive disturbance. Early stages may be febrile or afebrile. Cases take a rapid (acute and sub-acute) or slow (chronic) course. When death fails to end the disease the child lives as a subnormal child for a couple of years after clinical cure.

**The early stage:**—The playful and attentive infant becomes dull and apathetic and prefers to lie down on the bare ground. An earthy complexion, falling of hair from the scalp, sticky eyelids, commencing emaciation, wasting of the calf muscle, slight anaemia and digestive disturbances like anorexia, vomiting, constipation, bloating of the abdomen etc., slowly make their appearance. There is generally a low fever, and a few respiratory symptoms like cough and occasionally bronchial spasm (asthma-like).

The abdomen is generally protuberant due to intestinal distension. The liver is generally enlarged, soft, and painless. Spleen may or may not be enlarged. Examination of the chest reveals a few rales particularly over the right base, and weakening of the heart sounds. The tendon jerks are brisk, if not exaggerated.

Examination of the urine, motion, blood, and sputum may show nothing definitely abnormal. The urine on drying leaves behind a whitish deposit. (Case No. 83 studied in the three stages exemplifies the description in stages).

**The intermediate stage:**—The child at this stage has a liver up to the umbilicus which feels harder than before. The spleen may be palpable. In the strictly biliary type of cirrhosis, light tinge of jaundice, intermittent pyrexia and a characteristic cachexia start now. Either constipation or diarrhoea is present. The motion has lost its natural hue and odour; it generally assumes a whitish appearance, and has foul smell. Van den Bergh of blood is, according to results obtained by the writer, direct delayed positive. Urine is scanty, and is acid to litmus, and generally shows a trace of albumen and urobilin in excess. In the strictly portal type of cirrhosis, ascites is early in onset, and jaundice appears much later in the course of the disease.



FIG. 14.  
A female child aged 2 years (6-10-'44) with early hepatic cirrhosis. Enlarged liver is marked. (Case No. 83—1944)—I stage.



FIG. 15.  
(Case No. 83—1944). Advancing cirrhosis of liver. (Clinical photograph 5-1-1945).—II stage.

**The third or advanced stage:**—This is characterised by the presence of both jaundice and ascites. This has been described also as the terminal stage.

The liver is described to shrink as ascites develops; but the writer has failed to observe hepatic shrinkage in every case with ascites. Portal obstruction consequent on the shrinkage of the liver alone, cannot be held responsible for the development of the ascites. There seems to be other factors like toxic action and allergy.

"Ascites in cirrhosis of the liver may be due to hypoproteinaemia as well as to portal obstruction. It has been diminished in such cases by plasma transfusions and a high protein diet, which have no effect on the portal pressure."

In cases where the disease starts after a diarrhoea (generally in a summer) there is a marked preponderance in the enlargement of the

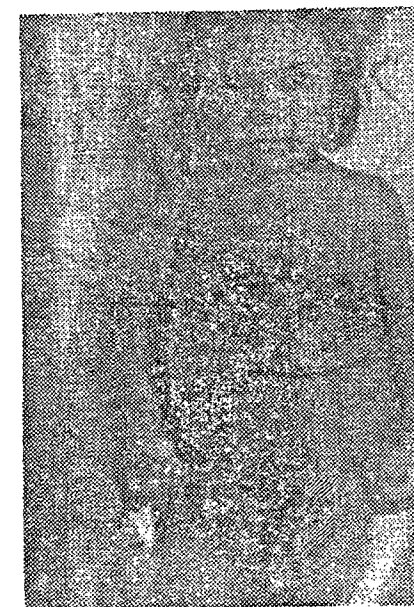


FIG. 16.  
(Case No. 83—1944). Very advanced cirrhosis of liver. (Clinical photograph on 20-1-1945).—III stage.

right lobe of the liver. The left lobe of the liver is found more enlarged in cases starting with chronic constipation. In all cases at this stage the spleen is definitely enlarged (1 to 2 inches below costal margin). It is the impression of the writer that children in whom the disease started with chronic constipation showed splenomegaly earlier in the course of the disease than in others.

In this stage, deepening jaundice, ascites, marked and continuous pyrexia, anæmia and a hæmorrhagic tendency are present. Micturition is scanty. Dropsy of the lower extremities appears in most extreme cases. Here again, toxic damage to the capillary endothelium and myocardial insufficiency are added to pressure on the renal veins within the abdomen to account for the dropsy. Distension of the superficial veins of the abdomen is characteristic now.

There is marked respiratory distress. This seems to be due not only to pressure of the ascitic fluid but also due to the damage of the lungs and heart which share in the common intoxication.

The right supra-clavicular fossa is found tender in some children with palpable lymph nodes also; and this point was stressed by the writer (1940).

Urine is scanty and high-coloured, and shows bile salts and bile pigments. *B. coli* may be isolated from the urine. Sputum does not show bacillus tuberculous. Blood examination reveals a secondary anæmia. Van den Bergh is direct immediate positive. Fragility of red cells is normal. The Kahn and Wasserman reactions of the child's blood or the mother's blood are negative. Motion is whitish in colour and with a foul odour. In a few cases thread worm or round worm ova are seen. The aspirated ascitic fluid is a transudate.

The long bones appear thinned out. There is clubbing of finger and toe nails in many cases. In a few children a downy growth of hair over the spine was evident. The skeletal muscles atrophy, particularly the calf muscles.

At this stage susceptibility to epithelial infections increases. Chronic rhinitis, chronic otitis, and chronically infected condition of the throat, tonsils and adenoids frequently co-exist.

A study of the common laboratory investigations is useful. The writer found large quantities of sulphur in the urine. Urine may contain sugar or sugar-like reducing substances. P. KRISHNA RAO (1934 and 1941) isolated pure cultures of *B. coli* from the urine in some cases. Faecal fat analysis is normal.

The hæmatological study by P. KRISHNA RAO (1941) showed a high total W.B.C. count, and differentially polymorph decrease and lymphocyte increase. Anæmia was not prominent. "Direct Van den Bergh test is negative, and indirect reaction is normal. In jaundiced cases the test is direct positive or shows a biphasic reaction, depending upon the intensity of jaundice, severe or mild."

The writer's investigations showed in the second stage of the disease a microcytic hypochromic anæmia, increase in the total white cells and differentially a relative preponderance of polymorphs and eosinophiles. In the third stage, the blood shows immature white cells and the coagulation time is increased.

The following table (*vide page 106*) summarises the blood findings in some cases in the writer's series.

X-ray study of the chest and bones, and electro-cardiographic tracings of a few children were done by the writer (1939).

*X-rays of chest*:—The general findings are (1) marked hilar shadows, (2) encroachment of right cardio-phrenic angle, and (3) shadows suggestive of involvement of the right supra-clavicular glands.

Electro-cardiographic tracings:—A.V.S. SARMA (1939).

Case No. 13:—Simple tachycardia. Deep Q in lead III. QRS of varying voltage in Lead III. T + + +. Tendency for flutter (?).

Case No. 14:—Simple tachycardia. T wave marked. QRS notched in Lead II. T + + +.

Case No. 22:—Simple tachycardia. Right ventricular preponderance. Deep Q. waves in Lead III. T + + +.

*X-rays of wrist*:—An arrest of growth, slight rarefaction of bone, and irregular epiphyseal lines are noteworthy. A few transverse lines of more dense osseous tissue are seen parallel to the epiphyseal line (Fig. 17). The writer suggests an analogy to the bony changes in renal and coeliac rickets.

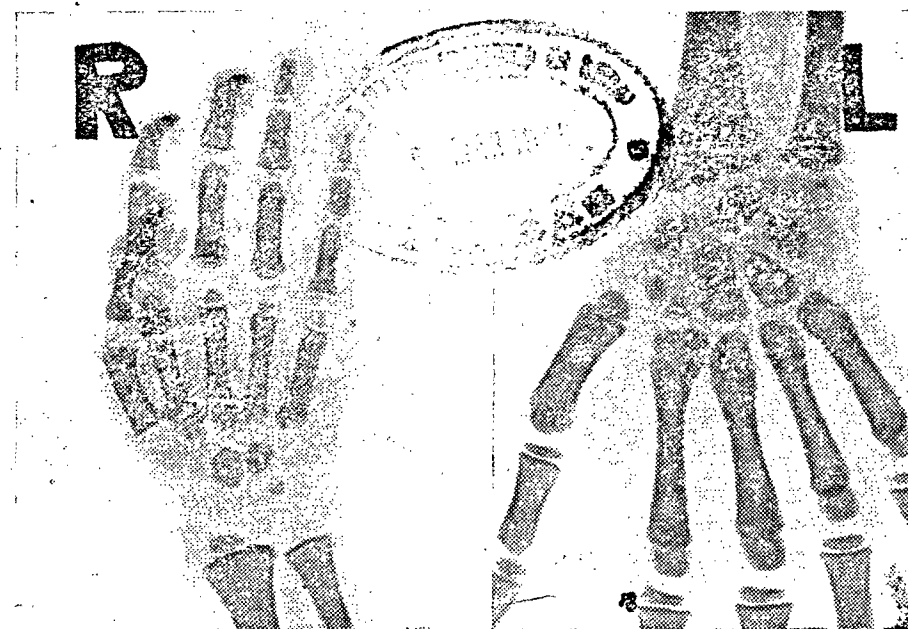


Fig. 17.

X-Ray wrist. Case No. 83 (3-1-1944).

The rarefaction of bones in general as revealed by X-Rays of bones was noted by K. C. CHAUDHURI (1944). The writer feels that because of hepatic cirrhosis the vitamins A and D are needed in larger quantities, and bones will certainly suffer from vitamin D deficiency in untreated cases of cirrhosis of liver in infants and children.

The Takata-Ara reaction in cases of infantile hepatic cirrhosis is positive (M. N. Pai 1941).



Agglutination of serum against dysentery group of organisms is negative.

**Metabolic disturbances:**—A study of liver efficiency reveals a few points. BHATTACHARYA and K. C. CHAUDHURI (1933) noted "the rise of blood sugar level after the administration of 10 gm. of lævulose by mouth amounted to as much as 131 mg. per cent. over the initial blood sugar level." The same has again been noted by K. C. CHAUDHURI (1944). These are due to the inability of the cirrhotic liver to produce and store glycogen.

Blood sugar is low in cirrhosis babies and particularly so at the third stages. Blood urea is not raised (P. KRISHNA RAO, 1941). The coagulation time of blood is delayed according to some writers, and the water metabolism is upset.

**Course of the disease.**—An acute case lived only for two months after the disease was first noticed showing early symptoms like constipation, fever and cough. A chronic case ran a course of more than 2 years and resulted in a "cure."

Children improperly treated or left untreated get progressively worse, and generally die of a complication like broncho-pneumonia, multiple abscesses and fever, or cholæmia, or die of gastro-intestinal hæmorrhage. While early cases have the best chances to recover, those in the late stages do claim rational treatment.

**Diagnosis.**—TIRUMURTHI and RADHAKRISHNA RAO in South India stress on the painless enlargement of the liver, peculiar intermittent pyrexia, earthy complexion, obstinate constipation, increasing icterus and portal obstructive signs and splenomegaly as salient features in the diagnosis of infantile hepatic cirrhosis. PANDALAI felt and rightly so, that the diagnosis is rarely made in the early stages.

Since the early symptoms go unnoticed and hepatomegaly is painless, most cases are missed until the cirrhosis is so advanced as to produce a protruberant abdomen, or decompensation has set in as evidenced by ascites. The ascitic stage is characteristic and diagnosis is easy at that stage.

Studying from the view point of febrile attacks, pyrexial conditions like malaria, kala azar, certain blood diseases, tuberculous peritonitis, hepatic tuberculosis, influenza and typhoid fever have to be remembered. In early stages where diagnosis is difficult, a trial treatment with Neostibosan may be tried as a therapeutic test. Blood examination generally excludes malaria and kala azar. Examination of the motion and sputum (obtained by gastric lavage) helps to eliminate worm infestation and tuberculosis respectively. The anterior margin of the liver in infantile hepatic cirrhosis seems to present a characteristic wavy border and there is always a preponderance of one lobe or other in the enlargement. This latter inequality in the enlargement of the liver, which the writer has observed, seems to be characteristic in this disease. This also aids in the differential diagnosis. In splenic anæmia (Banti's disease) the spleen enlarges earlier than the liver, and the jaundice is less intense than in hepatic cirrhosis proper. Splenic anæmia of infants (Von Jaksch's anæmia)—a condition fast disappearing from recent books on children's diseases—is characterised by considerable splenomegaly, absence of jaundice, and a blood picture of anæmia with myelocytes. Familial acholuric jaundice is spotted by family history, increased fragility of the red cells (sphyrocytes), absence of bile in urine, and presence of urobilin in large quality.

Gaucher's disease, Niemann-Pick's disease, Hand-Schuller-Christian syndrome, Von Gierke's disease and blood dyscrasias may also have to be remembered to complete the possibilities in differential diagnosis.

Rickets, hypertrophic biliary cirrhosis (Hannot), and conditions where jaundice is present, *i.e.*, yellow fever, Werb's disease, acholuric jaundice, congenital biliary cirrhosis and catarrhal jaundice, have also to be eliminated.

The enlargement of the liver in congestive heart failure, fatty and amyloid degeneration, tumours and cysts (echinococcus) should be remembered. The practitioner should also remember that simple soft non-cirrhotic enlargement of the liver is common in most children suffering from intestinal infections such as acute and chronic diarrhœas, dysenteries, etc. These conditions are amenable to treatment and the hepatic enlargement disappears with a cure of the intestinal condition. Ptosis of the liver is detected if the child is examined in both erect and recumbent postures.

The one condition difficult to eliminate is the enlarged liver of congenital syphilis. Apart from the serological tests, treatment with Mercury or Stovarsol will also serve as a therapeutic test when judiciously applied. In cases of congenital syphilis, ascites is rarely present, and jaundice, if and when present, is very slight. Radiologically the clavicular sign and the nibbling away of the upper and medial aspects of the tibial are characteristic of congenital cues.

Every medical student is aware of the fact that upto two years of age the liver is normally palpable below the costal margin. The Writer (1941) considers the following points as reliable diagnostic criteria for early hepatic cirrhosis:—

(1) The body weight of the child is lower than the normal average for the age.

(2) Gastro-intestinal complaints, particularly "cyclic diarrhœa" (A. V. S. SARMA 1939), disturbances like irritability, fever, constipation, torpor, cough, lying down on the ground, etc.

(3) Occurrence of an asthma-like syndrome after intake of certain foods or medicines—castor oil has been observed to produce a condition indistinguishable from asthma of allergic origin.

(4) Occasionally a vascular webby appearance is seen when the upper anterior abdominal surface is inspected and this is similar to the characteristic vascularity of the head of a congenital syphilitic child. A bulging of the umbilicus is also very characteristic.

(5) Enlargement and hardening of the liver and also of the spleen in advancing cases.

(6) The liver of children has to be felt very carefully and an outline may be marked with coloured pencil. The anterior margin of the liver is wavy or at least definitely irregular. The right lobe of the liver is more enlarged than the left in cases arising after the incidence of dysenteries or diarrhœas.

(7) Negative laboratory findings, which are of no diagnostic value.

(8) The urine of children developing cirrhosis and liver shows increasing quantities of Sulphur even on a fixed dietary. To demonstrate this the following procedure has been adopted by the Writer (1939):—To 2 c.c. of urine ten drops of 40 per cent. Sodium Hydroxide are added, and the whole is boiled. To it is added a small quantity of lead acetate. The

test is positive if the fluid turns intensely black owing to a precipitation of lead Sulphide.

(9) A critical study of the complete case-history in each case has been very useful in the writer's practice. The writer has often correctly anticipated hepatic cirrhosis from a study of the family history. Great importance is attached to the presence of allergic disturbances in the patient or his parents or his kith and kin.

**Prognosis.**—The clinical condition of infantile hepatic cirrhosis appears to embrace different types viewed from the point of pathogenesis. The probable types may be classed under the following heads:

1. Cases with allergic disturbances in the patient or in the family.
2. Cases where toxic and infective states (pre-natal, natal or post-natal) have an important bearing.
3. Cases arising mainly from dietetic errors.
4. Cases of obscure or mixed origin.

Prognosis can be assessed only after the origin of each case is determined. Rational treatment pays here. Prognostic decision has to be guardedly arrived at, as by the time clinical symptoms are clearly manifest in a case, enough damage to the hepatic parenchyma has already occurred and reparative physiological and immunological forces more or less exhausted. The softer and smaller the liver is, the better the prognosis. Children developing the disease due to an allergic diathesis have always good chances of recovery. Bilateral allergic inheritance is of worse prognosis than unilateral inheritance. The earlier the allergic manifestations (eczema, asthma, "cyclic diarrhoea") and the more they are in number, the worse is the prognosis. Bacterial infections add fuel to the fire of allergic diathesis.

Children developing the disease as a result of preliminary intoxication from the mother who has suffered from pregnancy toxæmia fare rather well with adequate treatment.

Ascites and jaundice mean a merciless march of events towards cholæmia and death as exemplified by cases 64 and 65.

Diseases like malaria, influenza, kala azar, whooping cough, eruptive fevers, etc., visiting a child with hepatic cirrhosis adversely influence the course of the disease.

The writer has noted that influenza leads to a greater enlargement of the liver and spleen, and increased cardio-pulmonary involvement. The onset of jaundice is to be dreaded.

"If the child has survived 3 years, the prognosis is good"—(M. B. PRABHU). K. C. CHAUDHURI gives a mortality percentage of 66 in his series. VAIDYANATHA IYER records cure in 26 out of 59 cases. MALLICK gives 2 per cent. recovery, while K. C. CHAUDHURI has 25 per cent. cures. In the writer's experience mortality is 80 per cent. and only about 20 per cent. get cured.

#### Part IV

**Treatment.**—A line of treatment for infantile hepatic cirrhosis is drawn out below, and the same is based mainly on personal study.

**PREVENTIVE TREATMENT:**—This part justifies elaboration as the curative treatment of advanced cases yields unsatisfactory results. This section is dealt with under the following heads: (1) allergy; (2) prevention of toxic and infective conditions; (3) diet and environmental care; (4) detection and treatment of early cases.

1. **Allergy:**—The writer believes that allergy forms the basis of a definite type of infantile hepatic cirrhosis. While the inheritance of allergic diathesis cannot be prevented, consanguineous marriages are to be avoided.

Mothers with allergic disturbances must control their diet during pregnancy and during lactation may have with benefit Pepsin and dilute Hydrochloric acid by mouth.

Ideal infant feeding means breast milk or in its absence acid milks with an optimum supply of vitamins A, B factors, C and D. Substances known to upset a baby (cow's milk or its rich fat, rice, potato, wheat, oils, castor oil, etc.) must be avoided. Unwary dietetic regime during an intestinal catarrh easily starts food allergy. Routine skin testing may be employed with standardised extracts of allergens and after detecting the specific allergens thereby, regular desensitisation, enteral and parenteral, may be employed both in the prevention and treatment of food allergies in infants.

Coming to bacterial allergy, avoidance of vaccination with vaccinia in a susceptible child is advisable. Wearing a mask by nurses or mothers who have upper respiratory infections is useful. Treatment of focal sepsis, and preventive measures against enteric infections, measles and pertussis are useful. Careful ante-natal care is essential, particularly in advising a balanced diet to the mother.

2. **Toxic and infective conditions:**—The various items that deserve study in this connection are: (1) toxæmias of pregnancy, particularly hyperemesis, eclampsia, anæmias, and neuritis; (2) constipation or diarrhoea in infants; (3) prolonged use of castor oil, chloroform, Chloral Hydras, etc., in pediatric therapy; (4) pertussis, diphtheria, typhoid fever, bacillary dysentery, repeated upper respiratory infections, sore throats including tonsillitis, small-pox, etc.; (5) helminthiasis.

The prophylaxis of toxæmias of pregnancy is not dealt with here.

Efficient treatment of constipation or diarrhoea is of paramount importance. Castor oil in large doses for constipation, is to be used with care. Treatment of diarrhoea (dietetic medicinal or specific) must be on sound lines to prevent later hepatic damage or allergic explosions.

The use of chloroform or chloral hydras is sometimes followed by damage to the liver. A child receiving chloral hydras, chloroform or their derivatives has to be carefully watched. A safe clinical method would be to administer such drugs for a definite period only. Chloral hydras or chloroform with or without the addition of codeine used in a proprietary medicine and administered to a child with pertussis for one month resulted in hepatic enlargement. On withdrawal of the recipe the child recovered.

Prophylaxis of bacterial disease is a subject that has to be strictly enforced in pediatrics. Preventive vaccination against small pox though of established utility in general, should be withheld in the case of a susceptible child—i.e., a child in a family wherein children are reported to have died of hepatic cirrhosis after vaccination.

Hygienic feeding of infants and efficient treatment of pica go a long way to prevent the incidence of helminthiasis. When worm infestation is observed, specific but safe treatment only need be given. Anthelmintics chosen should be non-toxic to the liver. (e.g. Helminal).

The existence of hepatic cirrhosis seems to be independent of concurrent tuberculosis and congenital syphilis. Incidence of tuberculosis, more commonly pulmonary tuberculosis, in a child with hepatic cirrhosis, only hastens the end of the child. A combination of congenital syphilis and hepatic cirrhosis carries much worse prognosis than either of the two. One such child under the writer's care died of ascites, jaundice, and cholæmia like any other child with advanced cirrhosis of liver.

3. *Diet and environmental care*:—Mother's milk is ideal for the baby and therefore breast-feeding should be strongly recommended. It is the general law of nature. The cow's milk is for her calf. Man stands between carnivora on one side and the herbivora on the other, viewed from the standpoint of intestinal digestion. Therefore even when artificially and ingeniously altered, cow's milk or goat's milk is primarily meant for the particular young one of the species which is purely herbivorous.

The technique of breast-feeding must be willingly mastered by every mother. Allergic mothers may take during the lactation period pepsin and hydrochloric acid with some advantage, and should always avoid things known to upset them or their children. A tuberculous or pregnant mother should stop suckling her child. Breast milk of a mother who had eclampsia may be denied to her baby. When a wet nurse is employed one has to remember occasional intolerance (anaphylaxis) from the baby's side.

Artificial feeding is the only choice open when there is failure of breast-feeding. Artificial feeding with cow's milk is a method which has to be mastered both by the mother and the nurse. There are recognised methods of artificial feeding:—(a) By determination of the percentage of the contained food elements, (b) by establishing the caloric value of food elements and (c) by reckoning the amounts of milk and water per pound of body weight in the light of accepted standard (PATERSON and SMITH emphasize the method as the simplest and most useful one). Goat's milk is also a substitute for breast milk. Some authorities prefer, and probably with ample reason too, a wet nurse in all familial cases.

When proprietary foods are used the usual scheme of a half cream dried milk till the end of the third month and full cream dried milk till the sixth month has to be modified in our country. It is better to give only half cream dried milk till the end of the sixth month. Intolerance to fat or protein of cow's milk has always to be borne in mind, and if intolerance is detected suitable proprietary products like Frailac, Lacidac, Almata etc., are used; but acidified milks or buttermilk suitably diluted may be used. It is essential to satisfy the buffer value of cow's milk by the addition of dilute hydrochloric acid.

In the method based on milk and water requirements per pound of body weight or on the addition of water to proprietary milk products, the writer feels that it is better to give concentrated feeds *i.e.*, by decreasing the diluent, and also to supply the baby additional drinking water in between the feeds. This slight adjustment has played a useful part in minimising the risk of vomiting and diarrhoea in infants. This concentration of foods is important in the feeding of infants with hepatic cirrhosis, as the water metabolism is deranged in the disease.

During the third quarter of an infant's life, weaning is attempted, and this process of weaning deserves great attention. Introduction of extra carbohydrate may with benefit be in the form of dextrimaltose (Karo syrup)

and not as pure starch (rice, sago etc.), which is the usual practice. Malt from cholam (great millet) promises to be a good substitute. The composition of malt from cholam is thus: Moisture 7.69%; ash 1.53%; crude protein 0.30%; fat 3.65%; crude fibre 0.31%; carbohydrate (by difference) 84.2%—total 100%.

Pungent, irritant, fibrous and non-nutritious elements in diet have to be eschewed to prevent gastro-enteritis. Administration of sweets and dhall pudding is only asking for digestive upset and food allergy. The dietary combinations in general are a legion, and the fond mother out of fondness and ignorance forces her baby in spite of his dislikes and protests to partake of items of her own diet, and this is the beginning of the trouble. It is therefore useful to educate the mother to respect her baby's dislikes and protests and not to disobey them, as the baby's instinctive dislikes seem to serve a protective purpose.

The diet should be sufficiently rich in calcium and phosphorus as also vitamins A, B factors, C and D. B<sub>1</sub> deficiency has been reported to produce adrenal hypertrophy, fatty degeneration of the liver, intestinal engorgement, and allied conditions. Vitamin C is in the writer's opinion an important dietetic agent for the prevention of infantile hepatic cirrhosis. It seems to exert anti-allergic, anti-marasmic, and hepato-tonic actions, and supports capillary endothelial integrity. "Treatment with vitamin C is rational as it has a direct action on the endothelial integrity of the hepatic capillaries and the essential lesion of the infantile hepatic cirrhosis is endothelial damage resulting in swelling and spasm with later fibrosis of the hepatic venous terminals" U. S. RAO (1940). Fruit juices (tomatos and oranges) are useful sources of vitamin C, but if children are sensitive to them, ascorbic acid should then have to be given. Ostomalt, Radiostoleum emulsion with vitamin C, Malt Extract with shark liver oil, are useful poly-vitamin tonics in pediatric therapy. The albumen of egg is probably ideal amino acid assortment, and may form an ideal dietetic adjuvant.

The later half of the first year seems to be the elected period for the clinical recognition of hepatic cirrhosis, and therefore that particular period has to be watched with care for an enlarging liver. Some authorities believe that the disease is detected mostly between 1 and 2 years.

Association of children with others having contagious diseases or frequent upper respiratory infections is undesirable. Water given to the babies needs to be clean and even sterile-sterilised by boiling.

Recent ideas on prevention of hepatitis are these:— "Experimental work carried out from a variety of aspects shows that while restriction of fat is indeed essential in conditions of strain or damage to the liver restriction of protein is not merely unnecessary but positively harmful.....Most of the recent work has been done on white rats, which develop fatty livers and eventually cirrhosis when they are fed on diets very rich in fat. Fatty degeneration can be prevented not only by choline but also by cascin, methionine and choline-like amine, betaine.....protein acts by virtue of its amino-acid (methionine).....A second important factor for the integrity of the liver is on unidentified component of the vitamin B complex which is present in yeast.....molasses may have a like protective action.....methionine is evidently a key substance in the metabolism of the liver." (*British Medical Journal*, 12-12-1942).

4. *Detection and treatment of early cases*:—The treatment of an early case is based on the following lines:—

Suspicion of cirrhosis in the baby is no indication, according to the writer, for the cessation of breast-feeding when the mother regulates her diet and preferably takes also an acid and pepsin mixture during the lactation period and thereby prevents the excretion of protein intermediaries into the breast. In case of artificial feeding, correct adjustment of diet, particularly decreasing of fat which is badly tolerated as a rule, increasing of carbohydrate—addition of ordinary cane sugar or glucose if need be—and giving raised protein of high biological value. Each feed is given preferably concentrated to some extent, and drinking water is given in between feeds. In the case of older infants *i.e.*, for those after the ninth month, increased carbohydrate is allowed. Well boiled congee of parboiled rice, honey and stewed fruit are allowed.

Addition of vitamins A, B factors, C and D and ensuring calcium and phosphorus in adequate quantities are important. A dietetic corrective like Ostomalt is welcome. Some children need specially large quantities of vitamins C and B factors, and in such cases Cevibex tablets may be prescribed. A good extract of yeast is desirable dietetic adjuvant.

“On a dry-weight basis yeast contains some 50 to 60% of protein—far more than any animal source—and this is stated to be as good biologically as the protein in milk. No animal can rival yeast in the proportion of protein it holds in its tissues. Yeast contains the essential amino acids tryptophane, methionine, valine, leucine, isoleucine, histidine and lysine. It is an excellent source of the B vitamins, and has nearly 2% of potassium, 5% of phosphorus, and 0.03% of iron, essential constituents of the diet. Among other important substances in yeast are glutathione and ergosterol (both 0.5 to 0.6%), glycogen (40%), lecithin (2 to 4%), and nucleic acid (26%). If yeast is irradiated the ergosterol is transformed into calciferol equivalent to 3,000 to 4,000 I. U. of vitamin D per gramme of yeast. A fifth of a gramme of irradiated yeast a day would thus suffice for the prophylaxis of rickets in the young infant.” (*British Medical Journal*, December 16, 1944—page 797).

Ensuring proper bowel action daily by the use of drugs like Milk of Magnesia, rhubarb, or cascara sagrada is necessary. Purgation induced by salts seems to predispose to urinary infections. The juice of leaves of *Eclipta Erecta* with equal quantities of honey was tried by the writer as a useful procedure.

A mixture containing pepsin and dilute hydrochloric acid before feeds or the use of acidified milks or lacidac in cases with allergic diathesis is desirable. Papain also has been used as an alternative to the above procedure.

A special treatment is the use of adrenal cortex orally or parenterally.

If the child does not improve and weight is stationary or is decreasing, vigorous treatment must be launched. Loss of appetite is a grave prognostic sign, and is ideally treated by infections of vitamin B<sub>1</sub>. The presence of normal appetite is a favourable point.

(To be continued).

## Acute Ulcer caused by Gram-negative Fusi-form Bacilli\*

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THIS special type of ulcer is met with in some of the Western districts of the United Provinces. This kind of ulcer is quite prevalent at Najibabad during the month of August, September and October generally. It has come to my notice since last year only; and I have been trying to investigate the nature and treatment of it since then. I am not aware if any of my medical colleagues has investigated and reported about this specific infective ulcer. My findings about this ulcer are as follows:—

**Seasonal prevalence and location of ulcer.**—Found almost exclusively during and after the rains till winter gets severe. In all my cases (numbering 33) the ulcers were found located on the lower extremities alone and in no other parts of the body. This specific ulcer is found among males and females alike and in persons of both clean and dirty habits. It is common both in the town as well as in the village. No age is exempt; but children seem to be affected more. The infection is commonly found to be superimposed on a simple wound or it starts anew as a pimple and then develops to a pustule simulating scabies, ultimately ulcerating into ugly ulcers.

**Character of ulcer.**—Small ulcers have a punched out appearance whereas single, extensive ulcer may have everted skin edge. Without exception the ulcer is found covered up thoroughly with thick, mucopurulent discharge having the colour and consistence of dirty cream. The discharge has practically no foul odour and is alkaline in reaction. The ulcer can easily be cleared off the discharge by means of soft dry gauze. No membrane or slough is noticed covering the surface of the ulcer. Base of crater of the ulcer is coarsely granular, pale in colour and does not bleed easily. It is a noteworthy fact that unlike other acute ulcers, there is hardly any active and visible tissue reaction around the ulcer, *viz.*, hyperæmia, induration and swelling barring a slight turgidity and tumidity of the surrounding tissue. Because of this feature the surrounding tissues are soft and not quite tender to touch. A peculiarity noticed about this ulcer is that in neglected cases it is found to spread more by surface extension than in depth, whereas in treated cases especially where it is bandaged tight, the ulcer increases more in depth than surface.

**Clinical features.**—In cases with extensive or multiple ulceration there is noticed a rise of temperature ranging from 100°F. to 103°F., with all the toxic symptoms, *e. g.*, feverish look, dry tongue, scanty and highly coloured urine, thirst, distaste, malaise etc. In mild cases receiving prompt treatment such febrile reactions are rare. In no case, however extensive or multiple the ulcer is, there has been noticed any enlargement of regional lymph glands draining the ulcerated area.

**Laboratory findings.**—Bacteriological examination of the discharge reveals only gram-negative, slender, fusiform bacilli in abundance with only stray pairs or tetrads of pneumococci occasionally. A fact worthy of note is that the discharge obtained from grossly neglected and exposed ulcers (cases coming from rustic villagers) even, do not contain any other pus forming cocci or bacilli. Spirochaetes also are absent. Some of these fusi-form bacilli bear spores on one side of their body and not on their

\* Specially contributed to THE ANTISEPTIC.

extremities, I mean the arrangement of spores is not terminal. Both the body of the bacilli and their spores are stained well with I in 10 carbol fuchsin preferably heated. The body of the bacilli and the spores as well are gram-negative. The discharge, ofcourse, contains abundant pus cells. Examination of blood from cases having ulcer for more than 5 days and running a minimum temperature of 99°F. reveals marked eosinophilia ranging from 12 to 20% and a little lymphocytosis averaging 25% as well. There is no increase in the neutrophils either with filamentous or entire nuclei.

**Treatment.**—This is the most important point for a practising doctor, and all the same it had been a great problem for me for sometime how to cure these apparently simple ulcers quickly and with cheap remedies till I could find out a very cheap and efficacious solution for local application. I have not tried any remedy by mouth or by injection excepting a brisk purgative and Sulphonamide. I have, so far, not received happy response with Sulphonamides by mouth. Blood report also does not indicate its use, I think, as there is no neutrophilic reaction. Besides the usual cleansing antiseptics *e.g.*, Hydrogen Peroxide and Electrolytic Chlorine or Eusol that I use as a routine in almost all types of wounds and ulcers, I have tried the following remedies one after another with no convincing success *e.g.*, 4% Silver Nitrate Solution, Copper Sulphate crystal, saturated solution of Magsulph in Glycerine, 1:6000 Hydrarg perchlor lotion, Ung. Hydrarg Co. Ung. Zinc Boric and Acriflavine. The only remedy that has given me a satisfactory result is the following solution.

|   |                 |     |        |
|---|-----------------|-----|--------|
| R | Zinc Sulph      | ... | grs. v |
|   | Copper Sulph    | ... | grs. x |
|   | Phenol          | ... | ℥ x    |
|   | Glycerine       | ... | 3 iii  |
|   | Aqua distillata | ... | 3 i    |

The ulcer is cleansed first with Hydrogen Peroxide followed by electrolytic Chlorine lotion, mopped dry and then gauze-soaked thoroughly with this solution, is applied on the ulcer and a loose bandage applied on. It is highly desirable to change the dressings twice a day as the discharge is generally profuse. Hot Mag Sulph fomentations applied directly on the ulcer decidedly hastens the process of healing and relieves pain afterwards. Dry heat applied over the dressing by means of a slow glowing fire beneath the cot while the patient lies down is welcome for the patient and his ulcer. This treatment shows a definite and appreciable change in the condition of the ulcer after 3 or 4 applications only and patient's agony of pain is relieved almost completely after the very 2nd application. When the discharge lessens considerably and the base of the ulcer turns into reddish pink from its original pale hue, this solution is found to cause some burning pain. Then, 10% Chlorbutol in almond oil or olive oil is painted over the ulcer and Thymol Iodide is dusted on it and dressed dry for about one or two days. Then again we can preferably change on to the same solution for quicker healing. As the character of the discharge changes from purulent to mucoid one and healthy granulations begin to appear (as manifested by the latter oozing blood on light moppings) 5% scarlet Red ointment is rubbed on the base and margin of the ulcer to stimulate their growth. I have found, without exception, that such ulcers heal up quickly and completely without any surgical intervention under this regime of treatment.

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## Pneumonic Complications in Enteric\*

C. ADINARAYANA RAO, M.B., B.S.,  
Parvatipuram, Vizag. District.

FROM my experience of five or six hundred cases of enteric during the last fifteen years, I have noted that there are two very different kinds of pneumonic complications in enteric. The mode of onset, the main clinical signs and symptoms, the course, the complications and the ultimate prognosis in these two types of complications are so very widely different and so very striking as to draw one's attention.

The first group is pneumonia typhoid, single or double. In this group which is rare (I have come across only six cases so far) the onset is sudden usually with a chill (simulating malaria), high temperature  $103^{\circ}$ - $104^{\circ}$ F. in the first week, resp. 40 to 50 and pulse 100. I have noted that in these cases, the pulse rate is slow as compared to the temperature (usual enteric). But the pulse-respiration ratio is 1:2 or  $2\frac{1}{2}$  and not 4, as is typical of pneumonia. This showed that there is more toxæmia and that the consolidation is more in area, the usual signs of pneumonic consolidation like limited movement, dullness on percussion, no breath sounds, increased V. F., tubular breath sounds, with a few occasional crepitations, scanty expectoration, dry cough are all present. Later on small quantities of thick sticky sputum appears.

The peculiarity in these cases is that on the 7th day the temperature comes down by crisis to normal or sub-normal (noted in some cases). My surprise is that the temperature of the enteric which is in the first half of the second week is brought down to the sub-normal by the crisis of pneumonia. The patient feels less toxic, happier and easier and there is an amelioration of all the pneumonic symptoms. That is as it should be in a case of pneumonia. But after 24 hours the temperature gradually rises and reaches the previous  $104^{\circ}$ F.- $105^{\circ}$ F. level in another 24 or 36 hours. What has become of the enteric temperature during the 24 hours (there is no hæmorrhage or any other intestinal complication to account for this fall. Every evacuation was regulary seen in these cases).

The temperature having risen to the usual enteric second week height, runs smoothly, cerebral complications are few, coma does not develop, myocardial weakness is not developed, the temperature touches normal by the end of the third week, and the patient is alright. He is none the worse for the early complication of pneumonia. Though at first the temperature is high, toxæmia is marked, and the general condition appears to be bad, after the pneumonic crisis, the picture changes and the patient has rest and runs a smooth course.

In the second variety, the more common group, there is consolidation of one lobe (usually the lower) of one lung as a complication of enteric. In these cases, the mode of onset is slow, there is no pain in the chest, mild cough and scanty expectoration are present. The temperature during the first half of the first week is  $103^{\circ}$ F.- $104^{\circ}$ F., respiratory rate about 25, pulse rate 100-110. I wish to emphasize here that the pulse rate as compared with the temperature is low (as is usual in typical enteric cases). The respiratory rate bears normal relation to pulse rate in the ratio of 1:4. In fact there are no rapid respirations compared to the temperature, no dyspnoea, and the patient has adequate rest. In fact unless a thorough

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and systematic examination is made, the consolidation is liable to be missed, and only the usual bronchitis is suspected.

This bright picture gradually becomes more serious in the second week and in the third week it is almost gloomy and during the fourth week it almost always ends fatally. There is no crisis on the seventh day (as is observed in the other group). The temperature continues to rise in the second week 104°F-105.5°F or 106°F. In the third week it reaches to 107°F. and the patient dies of hyperpyrexia (cold sponging, ice sponging continuously for an hour or at intervals, iced-enema, wet blankets, Cryogenin and Pyramidon have all been tried). They do bring down the temperature; but the effect is only transitory. Even in cases where after one hour's continuous iced sponging temperature was brought down from 105.5°F. to 103°F. it again rose up to 105.5°F. in another hour. After repeated spongings over some days, usually first half of the third week, the tension of the pulse decreases after each sponging, it sometimes disappears completely at the wrist during the sponging, but reappears after the sponging is stopped and temperature rises up. I do not suggest that the continued sponging is the cause of the failure of the pulse. I think, rightly or wrongly, that the temperature acts as a stimulant to the myocardium or perhaps the toxins causing both temperature and toxæmia act as a stimulant, and when the temperature is brought down the stimulant is no longer there and the tension of the pulse decreases. When the temperature increases, the pulse becomes perceptible gradually, the hyperpyrexia responds less and less to sponging and other methods, and auricular fibrillation sets in and the patient dies.

The cerebral condition in these cases is one of coma vigil. The eyes are open but the patient does not respond when called or questioned though loudly. The winking reflex is absent. The swallowing reflex is absent (Glucose water or milk poured into the mouth cautiously in small quantities is retained in the mouth, neither swallowed nor spat out). I had to do nasal feeding several times in many cases. Even 4 to 6 ozs. of coffee or milk causes difficulty and embarrassment of breathing due to myocardial weakness. There is incontinence of fæces and urine and sometimes retention. The patient dies in this stage of coma vigil.

There is no delirium—no talking at all—but in one case I have seen, an advocate aged 28 years, lecturing to me quite sanely and consistently in English without a mistake for 10 minutes, of course he would not stop nor answer questions nor respond. He died in 36 hours.

The skin in these conditions is cold and perspiring profusely. Surface temperature is normal but rectal temperature is 106°F. or 107°F., even the difference between rectal and oral temperatures is 2 or 3 degrees. Why this diaphoresis? Why the temperature continues to rise in spite of this very marked diaphoresis? Is it because the myocardium has been damaged very seriously, the arterioles have dilated completely, to allow the poor heart to work at a minimum pressure? Is it nature's own adjustment of the complex cardio-vascular blood pressure mechanism? The difference between systolic and diastolic pressures is very low. In such cases how can one justify the use of Coramine or other stimulants that can only stimulate a seriously damaged heart, make it overwork and fail all the more earlier! In fact I have not noted any effect, even temporary in such cases (Glucose intravenous, Berin intravenous, Oxygen inhalations have all

been tried with no success in these cases). I have committed the mistake of using Coramine in some of my earlier cases of this type, unknowingly at first, knowingly later, as it was the only and the last resort.

These cases of this first group are entirely different from those of the second group:—

- | 1ST GROUP.                                                  | 2ND GROUP.                                                      |
|-------------------------------------------------------------|-----------------------------------------------------------------|
| 1. Mild hyperpyrexia reacting to measures.                  | 1. Severe hyperpyrexia not responding to measures.              |
| 2. Delirium, wild or mild.                                  | 2. Coma vigil with no delirium.                                 |
| 3. Swallowing reflex present.                               | 3. Swallowing reflex absent.                                    |
| 4. No profuse diaphoresis.                                  | 4. Profuse diaphoresis.                                         |
| 5. Pneumonic crisis on the 7th day.                         | 5. No pneumonic crisis on the 7th day.                          |
| 6. Serious clinical picture at first but later improvement. | 6. Mild picture at first becoming more grave and serious later. |
| 7. Prognosis good.                                          | 7. Prognosis bad.                                               |

This consolidation of one lobe of the lung attended by a high respiratory pulse ratio of 1:2 with symptoms of pneumonia runs a mild course after a mild crisis. The other consolidation of the lobe of the lung attended by no symptoms of pneumonia and the high respiratory ratio of 1:4 with no crisis gradually becomes more serious and the patient dies.

What is the difference? I have been thinking that in the first group the pneumococcus is the cause of the consolidation and the infection must have been ordinary and not virulent or massive. It is only a superimposed pneumococcal infection on a mild enteric, sometimes in the first week, sometimes in the second week. Therefore it runs a smooth course after the pneumonic crisis. In the second group bacillus typhosus is the cause of the consolidation. The infection is virulent and massive, therefore there is no crisis and the prognosis is bad. The infection must have been heavy and virulent to have caused consolidation where bronchitis only should have been caused. Therefore it runs a malignant course. The complications are rarely intestinal. (In fact in all cases where there are severe intestinal complications like perforation and hæmorrhage, I have never seen this consolidation. The complications are only of the brain, lung, and heart, and not of the intestines.) Here are a few typical cases of both groups to emphasise my observations. Of the first group I am giving three case reports briefly out of the six as they are all typical.

*Case No. 1:—*Chinnammi, aged 25, H. F., seen on the 3rd day of continuous fever, consolidation of the base of the right lung with all the signs of pneumonia. Temperature 104°F., pulse 120, respiratory 45. On the 7th day the temperature came to sub-normal, the patient was conscious but as the body was very cold, pulse very feeble, and the eye sight vacant, nose and face sweating (signs of very bad prognosis in Ayurveda, she was placed on the floor, a custom prevalent among Hindus), there was the usual village hubbub, and when I rushed to attend upon her, I found that, but for the low temperature, everything else was not very bad, or hopeless. I persuaded her relatives to place her on a cot as the case is hopeful. I gave camphor in ether and 3 hours after, Coramine. The temperature was 97°F. for 36 hours. Next day at 12 noon it began to rise and gradually went up to 104°F. During the 2nd week she developed coma and I had to do nasal feeding for 2 weeks, 4 times daily. In the 3rd week she developed

higher temperatures 105°F. to 106°F. but always responding to ordinary measures. After the 21st day coma disappeared, temperature came down by lysis, and she was alright by the end of the 4th week. This was my first case, and I diagnosed pneumonia and thought the crisis was pneumonic. The subsequent rise in temperature and coma during the 2nd week made me think that it was enteric. When I observed the T. P. R. readings after the case was alright, I found R. : P. : : 4 : 1.

*Second case* :—Kondamma, aged 15, F.H., seen on the first day of fever, temperature 104°F. pain and other symptoms of consolidation of base of left lung present. Pulse 120, resp. 45. I subsequently found that she was exposed to the wet cold monsoon weather. I suspected enteric also because of the T.P. ratio. Crisis on the 7th day, temperature coming down to normal lasting for 24 hours. During the 2nd week hyperpyrexia upto 105.5°F. and semi-coma not requiring nasal feeding. She convalesced during the 4th week.

*Third case* :—Manmadharao, aged 20, H. M., fever seen on 5th day temperature 104.5°F. P. 115, R. 50, signs of pneumonic consolidation of left base, crisis on the 7th day, temperature rising up to 104.5°F. after 24 hours. Semi-coma during the 2nd week, temperature, 105 to 106 hyperpyrexia responding to ordinary measures. No need for nasal feeding, convalesced in the 4th week.

**In the second group.**—*Case No. 1* :—Sankara Sastry, H. M., aged 18, seen on 7th day of fever, consolidation of both bases right and left, temperature 105°F., R. 60, P. 150, very dyspnoic. There was no crisis in the 2nd week, temperature 105°F. to 106°F. body surface cold and clammy. On the 13th day temperature 107°F. and coma vigil. Patient died on the 13th day due to hyperpyrexia and auricular fibrillation. Efforts to bring down the temperature were useless.

*Case No. 2* :—Kasipathirao, H. M., aged 27, seen on the 3rd day of fever, temperature 103°F. P. 110, R. 30. Signs of consolidation of base of left lung, no crisis on the 7th day. 2nd week hyperpyrexia and semi coma, 3rd week hyperpyrexia coma vigil, and cold clammy surface of the body. The hyperpyrexia did not respond to any measure. Patient died on the 21st day due to hyperpyrexia and auricular fibrillation.

*Case No. 3* :—Desikachari, H. M., aged 28, seen on 7th day, temperature 104°F., P. 120, R. 30, consolidation and pneumonic signs of base of left lung. No crisis, during the latter part of 2nd week he developed semi-coma, hyperpyrexia and coma vigil, and cold clammy body surface, and died on the 17th day due to hyperpyrexia and auricular fibrillation.

**Summary.**—Pneumonic signs in enteric are common. In cases of superimposed complications of pneumonia on enteric either coming on the same day or slightly after there is a crisis on the 7th day of its onset. The clinical picture at first serious, clears up and the patient convalesces in the 4th week. This consolidation, I think, is due to pneumococcus.

In the second group which is more common the signs of consolidation and other signs of pneumonia are present. There is no crisis. In the second and third weeks hyperpyrexia, coma vigil and profuse diaphoresis set in, and the patient dies in the third week due to hyperpyrexia and auricular fibrillation. The pulmonary consolidation in these cases is due to bacillus typhosus group. Further research and observations are necessary to elucidate many points in this clinical report.

## Bacillary Dysentery\*

HARENDRA NATH BAGCHI, M.B.,

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It is an acute epidemic disease due to the invasion of the mucus membrane of the large intestine by bacillus dysenteriae characterised by sudden onset, frequent mucus and bloody stools, colicky abdominal pain, tenesmus and intense toxæmia terminating abruptly in death in 2 to 15 days generally in 2-3 days, or by gradual improvement or it passes into chronic stage.

**Pathology.**—By entry into the mucus membrane of the large intestine the dysentery bacilli multiply and liberate a toxin which on absorption and subsequent excretion causes an inflammation of the whole colon from catarrhal inflammation to complete coagulative necrosis. Hæmorrhages may occur in the mucus and sub-mucus tissues, the lymphoid tissues being also swollen. Later on there is an out-poring of mucoid and bloody exudate into the lumen of the bowel, the swollen mucus membrane shows areas of necrosis, the separation of which leads to superficial transverse ulcers, their edges are ragged and the floor is covered with greenish sloughs.

Anatomically by acute diffuse inflammation of the large intestine it leads to superficial necrosis of mucus membrane by hyperplasia and hæmorrhagic infiltration of lymph follicles, it causes induration and thickening of the intestinal walls, hæmorrhagic swelling of meso-colic glands and parenchymatous changes in the viscera.

**Bacteriophage.**—If dysentery dejecta are diluted with bullion cultures and then filtered and small quantity of filtrate is added to bullion culture of dysentery bacilli, the bacilli are dissolved. The action of this lytic agent was due to an invisible filterable virus which was able to multiply in the presence of the living bacteria. This bacteriophage appears in the 1st week of the disease and persists into 3rd or 4th weeks of the disease. Bacteriophage is a filterable substance which causes lysis, it increases in quantity and can be transmitted by one culture to another.

**Classification.**—1. *Bacillary dysentery* :—(a) Due to *B. dysenteriae*, Shiga; (b) due to *B. dysenteriae*, Flexnor; (c) due to *B. dysenteriae*, Sonne-Hissy.

2. *Protozoal dysentery*.

3. *Bilharzial dysentery* : (a) Due to *B. mansoni*; (b) due to *B. japonicum*; (c) due to *B. hæmatobium*.

**Etiology.**—It is prevalent almost all over the world, very cold countries excepted. It breaks out in epidemic form in boarding-schools, prisons, lunatic asylums, etc. Bacillary dysentery breaks out in early summer, rainy season and in autumn. Age—no age is immune. Children are liable to be attacked. Debility, low vitality, fatigue and chills, errors in diet and drink, chronic intestinal disorders and over-crowding predispose to it.

The dysentery bacilli gain entrance to the large intestine by the intake of contaminated food or drink by :—

(1) Carriers—e.g., cooks, servants who distribute food.

(2) Flies—flies carry faecal matter in their feet and contaminate food and drink.

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(3) Water-supply contaminated with faeces of patients. Incubation period—usually 2-4 days but it varies from 1-7 days.

**Types.**—1. *Mild type*:—Slight fever, stools 4-8 in number, liquid motion contains little blood and mucus, slight toxæmia—recovery in 5-6 days.

2. *Severe type*:—Sudden onset, fever remittent and moderately high, stools—15-25 in number, loose stool containing blood, mucus and pus, with no faecal matter, severe abdominal pain and tenesmus.

Marked toxæmia: (a) pinched faeces with sunken eyes (signs of dehydration); (b) tongue dry and coated with brownish white fur; (c) rapid thready pulse; (d) tenderness over the whole of abdomen; (e) intense thirst, vomiting, scanty urine and prostration; (f) moderate leucocytosis 12,000 to 15,000, relief of symptoms in 2-3 weeks.

3. *Fulminating type*:—Death occurs in 2-3 days—high temperature intense toxæmia with stupor, rapid thready pulse, signs of marked dehydration.

4. *Gangrenous type*:—Passage of gangrenous sloughs in stools having a stinking odour.

5. *Choleric type*:—Watery and sero-sanguineous stools, subnormal temperature; early symptoms of collapse. Death in 2-3 days.

6. *Chronic bacillary dysentery*:—Occasional diarrhoea and stools contain muco-pus, with symptoms of dyspepsia, emaciation, anæmia and oedema of the feet may be present.

**COMPLICATIONS.**—(1) Acute circulatory failure; (2) Broncho-pneumonia (3) Nephritis; (4) Arthritis mono-articular and poly-articular; (5) Eye complications—acute conjunctivitis, iritis.

**SEQUELAE.**—1. Partial stenosis and dilatation of the bowel leading to marked constipation; 2. Chronic diarrhoea; 3. Symptoms of dyspepsia causing anæmia and emaciation; 4. Neuro-circulatory asthenia, palpitation, tachycardia; 5. Ascites due to a chronic periostitis.

**PROGNOSIS.**—Cases of Shiga infection if not treated early have a high mortality. Cases with marked toxæmia, continuous high temperature, numerous stools, hiccough, anuria and signs of marked dehydration have a bad prognosis.

**DIAGNOSIS.**—*Laboratory data*:—(1) Blood examination shows a moderate leucocytosis, with increase of polymorpho-neuclears.

*Stool examination*:—90% of degenerated polymorphoneuclear cells, about 4% macrophage cells with ingested red cells, ghost cells (*i.e.* shadows of polymorphoneuclears with cell contents thrown out). Red blood cells—unaltered and retain normal size. Intestinal epithelial cells in large numbers. Few bacteria.

2. *Stool culture*:—Stool should be fresh, uncontaminated with urine—a bit of blood-stained mucus be chosen; culture made in special media within 1-5 days. Positive results are obtained in 70% cases. Negative result does not exclude bacillary dysentery.

3. *Serum agglutination*—develops after 10-12 days and is not helpful.

**DIFFERENTIAL DIAGNOSIS.**—1. *From amœbic dysentery*:—In amœbic dysentery—Macroscopic features—colour—chocolate colour due to altered

blood, Reaction-acid, Microscopic features—Presence of *E. histolytica*, polymorphonuclears scanty about 7.5%, Macrophages absent, ghost cells very few, R.B.C. in clumps, presence of Charcot-Leyden crystals.

2. *From algid type of malaria*:—In a malaria very little blood in stool, icteric tinge of skin and conjunctiva, absence of much abdominal pain and tenderness, fever and dysenteric symptoms seldom appear together, spleen might be palpable. Detection, parasites in blood symptoms respond to quinine.

3. *Food poisoning*:—History of simultaneous affection of all persons after a feast, constant vomiting, absence of blood and mucus in stools, negative findings for bacillary dysentery.

4. *Cholera*—usually afebrile—stool watery, rice-watery with small flakes of mucus. No abdominal pain or tenesmus.

**TREATMENT:**—Conserve the energy of the patient by rest; (2) reduce toxæmia; (3) ensure rest to the bowels by suitable non-irritating liquid diet; (4) combat dehydration; (5) treatment of special symptoms and (6) specific treatment.

I begin treatment with Ol. Recini 3p with Tinopii m x added when no diarrhoea is present, followed by saline mixture the following 2 days. *e.g.*

|                |     |            |
|----------------|-----|------------|
| Sodi Sulph     | ... | 3 p        |
| Mag Sulph      | ... | 3 p        |
| Acid Sulph dil | ... | m x        |
| Aqua ad        | ... | 3 i t.d.s. |

**SPECIFIC TREATMENT.**—1. Sulfa-guanidine every 4 hourly for 4 days. Thereafter 1 tablet every 8 hourly for another 3-4 days.

• Plenty of water with Glucose D or Detrasal given by mouth as safeguard against renal dangers.

2. Sulfathiazole 4 tablets daily for 4 days and 3 tablets daily for 3 days.

3. Anti-dysenteric serum (Polyvalent) containing antibody against both Shiga and Flexnor bacilli and can be given without waiting for the bacteriological results.

Indications for serum treatment—(1) marked toxæmia and collapse; (2) high temperature; (3) severe abdominal pain; and (4) stools 20-30.

4. Desenteric vaccine (Flexnor) dosage 250, 500, 1,000 millions. I use 2 doses at intervals of 7 days.

5. Bacteriophage—ampoules of 2 c.c. t.d.s. in empty stomach for 4 days.

**TREATMENT OF SPECIAL SYMPTOMS.**—1. Kaolin with animal charcoal 3 tea-spoonfuls in water t.d.s. to combat diarrhoea, fever and toxæmia.

2. Targesin (colloidal albumin with diacetyl tannic acid and silver-salt)

3. Rivanal—0.1 grm. used to combat diarrhoea and toxæmia and to reduce the number of stools.

B. Relief of severe abdominal pain and tenesmus—(1) a flannel binder over abdomen; (2) hot water bag; (2) turpentine Hb stupes; (iv) suppositories of Morphia and Cocaine.

C. Collapse—Intravenous Saline cum glucose.

**Diet.**—In acute cases only administer boiled cool water for 12 hours in Shiga infection—administer warm albumen water, barley or arrowroot

water sweetened by lactose given frequently in small quantities—avoid giving milk.

In Flexnor infection avoid giving carbohydrates. Use lime-whey, chicken broth and raw meat juice.

In both infections apple-powder 4.8 per cent. suspended in boiled water without sugar may be given because pectin has got toxin absorbing power. Orange juice and baked bael fruit may also be given. When acute symptoms have subsided by the disappearance of mucus and blood from stools gradually add sago-puddings, milk puddings custard, and when the patient has been free from symptoms for 1 week allow soft rice, fish soup, boiled vegetables. Poached eggs may be allowed and *dahi* (curd). It is necessary to regulate diet for at least 6 months to prevent a relapse. It is better to give Berin (Glaxo) and Celin (Glaxo) 1 tablet twice daily for 1 week.

*Chronic cases*:—Allow bland non-irritating diet of high caloric value with fair quantity of protein, moderate quantity of carbohydrate and very small quantity of fat. It should be rich in vitamins.

I give soft rice, chicken jug soup, magoor fish soup, the fish to be only boiled, kach-kola made into paste with *dahi* and sugar added, lightly boiled goat's milk, orange juice, baked bael fruit and sugar, half of an apple fruit, banana and one pouched egg a day watching the stools carefully all the while. Cases treated by me—6 acute cases of bacillary dysentery treated by me with following symptoms, *viz.*, fever interemittent for 4-5 days ranging between 103°F. to 100°F., stools 20-25 per day with viscid mucus and bright red blood but no faecal matter, odourless, reaction alkaline, toxæmia, intense colicky abdominal pain and tenesmus, tenderness over sigmoid colon, tongue thickly coated with a brownish fur and dry tongue, pulse—rapid and thready.

Stool examination, R.B.C. (normal shape and size) macrophage cells, ghost cells, micro organism; reactions stools alkaline.

Blood examination, W.B.C. 12,000-15,000 (slight leucocytosis) absolute rest in bed, abdominal binder, bed pan, a nurse and boiled cool water in frequent sips for 12 hours.

*TREATMENT*:—Ol. Ricini 3 p.; Tin opii m x. stat.

In 3 cases—Sulfaguanidine 7½ grains tablet every 4 hourly for 4 days and every 8 hourly for 4 more days. Water to which Glucose D was added was given frequently to avoid acidosis and safeguard kidneys.

From 2nd day to 4th day of treatment I prescribed Sodi Sulph 3 i, Aqua Chloroform 3 i, t.d.s.

The Sulfaguanidine acts against bacillary dysentery due to its low absorability which causes it to remain in the colon to exert its bacteriostatic influence on bacillary dysentery.

Albumen water; barley water, both sweetened by lactose, tea without milk sweetened by lactose were given to the patients. Apple in powder suspended in water without sugar given to the patients because of the toxin absorbing power of Pectin and Cellulose, also orange juice, boiled bael fruit given. Berin and Celin tablets were given B.D., gradually sago-puddings added. On the 8th day soft rice, magoor fish soup, boiled vegetables given. Horlick's milk given from 2nd week.

In 2 cases of acute bacillary dysentery I treated with Sulfathiazole (marketed under the name of Thiazamide) giving 4 tablets daily for 4 days

and 3 tablets daily for 3 more days. Plenty of water with Dextrosal added, given by mouth. Diet as described above given also in these cases.

In one case with severe diarrhoea and signs of dehydration and collapse, there was occasional vomiting high fever ranging between 104°F. to 102°F., thready rapid pulse (130 p.m. and stools 30 in 24 hours. I gave anti-dysenteric Serum (Polyvalent) 50 c.c. intravenously and Normal Saline 0 i Soda Bicarb 3 i mix: sterilised by boiling injected intramuscularly.

2nd and 3rd days.—Anti-dysenteric serum 30 c.c. repeated given intramuscularly now.

Rectal saline with liquor Adrenalin Chloride 3 iv B.D. administered for 3 days. Kaolin 3-4 tea-spoonfuls in water given thrice daily for the first day of onset. All my cases recovered.

I treated 2 chronic bacillary dysentery cases with the following symptoms. Frequent passage of blood-stained mucus, patients afebrile but anæmic and emaciated. There were tenderness and thickening over descending colon.

Stool examination:—Red cells few, presence of mucus, pus cells few, macrophage cells nil, bacteria in abundance. Cultural examination in one case Flexnor bacilli isolated.

*TREATMENT*:—Flexnor Vaccine 250 mills. on 1st day and 500 mills. on 7th day. Ishapgulha seeds 2 table spoonfuls daily as laxatives. Injections Campolon and a mixture of Ferri et Ammon-citras gr. x to 3 i. t.d.s.

In the 2nd case—Bacteriophage 2 c.c. t.d.s. for 2 weeks and bowel lavage with Albargin gr. 1 to 3 i of cold distilled water. Cases recovered.

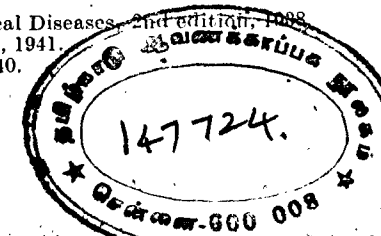
*Differential diagnosis between amœbic and bacillary dysentery.*

|                                                                                            |                                                                                                         |
|--------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------|
| " B. dysentery.                                                                            | Amœbic dysentery.                                                                                       |
| Acute disease with a tendency to epidemic spread.                                          | A chronic endemic disease.                                                                              |
| Incubation period 7 days or less.                                                          | Incubation period 20 to 90 days or more.                                                                |
| Onset acute, pyrexia common.                                                               | Onset insidious-pyrexia sore.                                                                           |
| Complications—polyarthritides and eye complications.                                       | Complications—hepatitis and other abscesses.                                                            |
| Death due to exhaustion, toxæmia.                                                          | Death due to exhaustion perforation, hæmorrhage, liver abscess.                                         |
| Stools—scanty, many in number, bright blood-red, viscid mucus odorless. Reaction alkaline. | Fæces intermingled with blood and mucus (anchovy sauce) very offensive generally copious reaction acid. |
| Blood examination—no leucocytosis in the early stages.                                     | A moderate leucocytosis is usual.                                                                       |
| Microscope—R. B. C. pus cells, Macrophage cells, micro-organisms.                          | R. B. C. in clumps entamœba histolytica Charcot-Leyden Crystals."                                       |

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## Burns and Treatment of Third Degree\*

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**Definition.**—A special type of an wound caused by action of dry heat to the body surface is known as burns. Broadly speaking it is produced either by direct contact with the flame, heated materials or by application of steam or hot liquid. The latter in general is known as scalds. The local history of burn is generally similar to the different stages of an ulcer, viz., stages of irritation, inflammation, sloughing and repair.

**Classification.**—The old popular Dupuytren's theory six degrees of burns is now replaced by one which consists of three degrees standing on the clinico-histological basis, viz., 1st degree—involving the superficial layers of the epidermis and forming erythema which is due to temporary contact with heated solid material or fluid much below boiling point of water; 2nd degree—involving the deeper layers of the epidermis causing vesication, yet in the layer of the stratum germinativum sufficient epithelial cells surviving which help the wound to heal quickly. It may be due to flame, highly heated material or fluid above 109°C. If it be due to flame there is blackening of the skin and scorching of hair; 3rd degree—involvement of both the epidermis and dermis causing death of the part without complete destruction of all epithelial elements and the blisters produced are ruptured and cracked. This is due to prolonged contact with flame or very highly heated substance or liquid.

**Signs and symptoms with the underlying pathology.**—Dealing specially with the *third degree* the most important symptom is hæmogenous shock, secondary one which, according to Kandrick, is the "clinical condition characterised by the progressive reduction in circulatory blood volume due to increased capillary permeability and with a continuously increasing tissue anoxia."

Clinically the onset of this secondary shock is represented by pale and collapsed condition with a clammy skin, a rapid thready pulse, increased respiratory rate and lowered B. P. Burn shock is accompanied by an electrolytic imbalance or acidosis, causes being dehydration and improper or absent kidney function.

The shock in burns is more pronounced by pain, fear, cold, exhaustion, plasma loss, and dehydration.

The chief pathological event is increase of R. B. C. count and hæmoglobin content, hæmococoncentration and fall of blood chloride. The toxic shock of burns is pronounced by absorption of specific toxins being produced at the burnt area or products of protein lysis caused by autolysis in the wounded tissues or by exposure to cold. The latter causes tissues anoxia which helps increased capillary permeability and plasma rich in protein pours out into the extra cellular spaces causing oedema.

Clinically this acute toxæmia is manifested by abrupt sharp rise of temperature, convulsion in children, nausea, vomiting, restlessness.

Finally the stage of repair depends on the virulence of sepsis, vitality of the patient and depth of area involved.

**Complications.**—1. *During the course of illness*:—(a) broncho-pneumonia; (b) meningitis; (c) stupor; (d) convulsion; (e) coma; (f) tetanus; (g) gangrene; (h) septic-endocarditis; (i) acute nephritis; and (j) toxic jaundice.

2. *Due to faulty technique of treatment*:—(a) contracture; (b) deformity; (c) ankylosis; (d) keloid body and scar formation; (e) palsy.

3. *Late complication*:—Acute duodenal ulcer—known as Curling's ulcer which is sometimes perforated causing peritonitis.

**Prognosis.**—Depends on: (1) degree of burns; (2) depth and area involved; (3) method of treatment; (4) sepsis and infection; (5) age; (6) general condition of the patient; (7) length of time exposed to burning agent; and (8) associated injuries to the vital organs.

**Causes of death.**—(1) shock; (2) asphyxia; (3) collapse; (4) septicæmia; (5) heart failure; and (6) intercurrent diseases.

**Treatment.**—In this article the general principle of the third degree burns is noted down and before going into detail it is absolutely necessary to record the following: (1) B. P.; (2) pulse rate; (3) Hb percentage; (4) hæmatocrit readings every 3 hours during the first day; (5) total quantity of R.B.C.; (6) graphic chart of total output of urine in 24 hours.

**Treatment proper.**—First aid—According to KOCH, S.L., a severe type of patient should be transferred immediately to an well-equipped hospital by covering the burned area with clean towels. There should be no temptation to treat him at home.

The main principles in the treatment are grouped as follows:—

- |                                                         |                                   |
|---------------------------------------------------------|-----------------------------------|
| I. The first and the foremost                           | III. Nutrition and fluid balance. |
| is the adequate treatment of shock.                     | IV. Skill and careful nursing.    |
| II. Care of the wound to prevent and control infection. | V. Late local care.               |
|                                                         | VI. Treatment of complications.   |

Anti-tetanic and anti-gas gangrene serum should be pushed I.M. as a routine treatment as soon as the patient is admitted into the hospital. Perfect rest with comfortable environment should be followed from the very beginning.

I. **Adequate treatment of shock.**—No local treatment must be undertaken till shock has been successfully treated. The following are the outlines to combat shock.

1. *To relieve pain*:—Morphine is the drug of choice and repute. The initial first aid dose is Morphine Hydrochlor gr. 1/3 cum Atropine Sulph. gr. 1/100 I.M. and can be repeated if necessary without least harm. But it is noteworthy (BEECHER H. K.) that one should not treat restlessness due to anoxia with Morphine as well as the dose must not be a high one for the latter brings the danger of respiratory depression. In case of urgency Morphine gr. 1/8 to 1/6 can be given I.V. with much precaution due to poor absorption from the subcutaneous route.

2. *To maintain body heat*:—By electric heat cradle, hot water bag, blanket. Over heating to be avoided.

3. *To treat shock proper*:—As stated previously that loss of extensive plasma is the chief cause of the shock so to replace the same is the chief motto of the treatment and is life saving. The quantity to be given

\* Specially contributed to THE ANTISEPTIC.

is estimated as follows: (a) Where no laboratory facility is available.—The quantity is based on the size and surface area of the burn (BERKOW, S.G.), or give 50 c.c. of plasma for every 1% of the body surface burned (HARKINS, H. N.); (b) if there be laboratory facility—Principal formula of HARKINS, H. N., LEE, W. E., and Black, D. A. K. can be adopted, viz.

- (i) 100 c.c. plasma for every point hæmatocrit exceeds normal of 45.
- (ii) 50 c.c. plasma for every point Hb exceeds normal of 100.
- (iii) 50 c.c. plasma for every 100,000 red count exceeding normal of 5 millions.

CANNON recommends that a pulse rate of 120 with a systolic pressure of 80 requires transfusion which should be as early as possible and adequate quantity to be given. When 1/6th to 1/8th body surface is involved with the above pulse rate and systolic pressure 1,000 to 5,000 c.c. of normal plasma is pushed in first 24 to 48 hours. Repeated hæmatocrit readings should be noted down to observe improvement by plasma transfusion.

Besides plasma the following can be used as substitutes for burn shock, viz., Isotonic solution of Sodium Lactate orally, gum acacia transfusion.

Adrenaline Cortical extract, Eucortine 2 c.c. I.M. every hour has been recommended but LEE and others are of opinion that this does not produce any satisfactory result.

Inhalation of oxygen should be carried on from the very beginning to check the pulmonary œdema and distress.

**II. Care of the wound to prevent and control infection.**—There should not be any initial cleansing and dressings for a few days in a well established shock. Simply cover the wounded part with sterile gauze. When the shock is over, with utmost gentle care and with full aseptic precautions the wound is cleaned. It is advisable to use anæsthesia in all extreme cases for which Sodium Pentothal I. V. is the ideal one. Locally a satisfactory cleansing agent viz., 10% Aqueous solution of Cetyl Trimethyl Ammonium Bromide (C. A. T. B.) can be used or simply soap and tepid water. Dead skin and ruptured vesicles are snipped off with sterile scissors and intact blisters are left. Lately the treatment of the wound after cleaning depends on two types of therapy, viz., (1) open therapy; and (2) closed therapy.

1. *Open therapy*.—Here with aseptic precautions one can dress the wound with drugs as often as necessary to clean it and can watch the degree of epithelialisation and improvement. But there is every chance of inviting secondary infection which can be controlled by local chemotherapy. After initial dressing keep the wounded part in a suitable position and ease with the help of splints so that no contraction and deformity takes place in future. The frequency of subsequent dressing depends on the general condition of the patient and the degree of infection of the part. The less the infection the less frequent the change of dressing. In the subsequent dressings all exudate is washed gently with Normal Saline and dead sloughs are snipped with scissors. To combat infection the patient is now treated with chemotherapeutic drugs both locally and by injection. Locally the wound is dressed with unguentum containing Sulpha drugs. Recently, Penicillin, the wonder drug, is added to the Sulphanilamide unguentum in strength of 100 units per gram. Besides the local application Penicillin is

pushed 10,000 to 12,000 Oxford units every 3 hours, total dose being about 900,000 Oxford units. If the wound is clean and locally no sepsis is apprehended it be simply dressed with gauze soaked with sterile Olive oil or Codliver oil.

2. *Closed therapy*.—The principle of this therapy is that the wound is treated with a chemical agent which is not removed frequently. By this invitation of infection from outside contamination is lessened but the main disadvantage is that the surgeon cannot often see the condition and rate of improvement of the healing of the wound. This therapy can be produced with two elements, viz., Chemical agents and Plaster of Paris bandage.

(a) *Chemical agents*.—Drugs used are either coagulants, e.g., Tannic acid, Silver Nitrate or dyes. The most important coagulant is Tannic acid. 10% of the solution is sprayed on the cleaned surface of the wound with a spray bulb. Allow it to dry till a firm coagulum forms. It is essential to take special care of the coagulum for cracks appear at the edges of the eschar which hold a foot-hold for carrying the infection. So these fissures should be sealed by cream of gentian violet or brilliant green. Recent advances of treatment with Tannic acid show that it does more harm than good. CANNON and COPE have proved that it is toxic and damages the epithelial cells. HARTMANN and ROMENCE have confirmed that Tannic acid causes liver damage and impair the liver function. Recent opinion is that Tannic acid application is not so much harmful on small burns but if 1/3rd of the body surface is burned it causes fatal liver damage by absorption from the tissues.

(b) *By Plaster of Paris bandage*.—(LEYENSON and LUND) The underlying principle and technique is the same as that of WINNETT ORR treatment in case of acute osteomyelitis.

**III. Nutrition and fluid balance.**—A patient of third degree burns develops loss of appetite due to toxicity, loss of plasma and dehydration. So it is essential to put him on high protein and vitaminous diet as cellular regeneration is favoured easily and rapidly by much protein and vitamin intake. At the same time it is noteworthy to maintain 3,000 C of which major portion should be protein. Give enormous fluid per mouth to obtain a free urinary outflow daily. Glucose 25% 50 c.c. with vitamin C is also to be pushed I.V. Vitamin B is also of much value.

**IV. Skill and careful nursing.**—Prognosis and rapid improvement of a severely burnt patient depends mostly on skilful nurse who should be smart, intelligent and sympathetic. Patient's morale to a large extent depends on a trained nurse. She who keeps her patient always cheerful, comfortable and knows all the procedures adopted in a well equipped hospital is an expert nurse.

**V. Late local care.**—Attention to be paid that in all third degree burns skin grafting should be done soon on the new resulting granulating area. The best time for grafting is just after subsidence of shock and toxæmic phases. Delay will lead to bad scars, deformities, contractures and disfigurements. Autodermal grafting is the best.

**VI. Treatment of complications.**—This is purely systematic and to be followed on the clinical manifestations during the course of treatment and afterwards as sequela.

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## Some Problems in Diagnosis of Pulmonary Tuberculosis in the Tropics

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BASED on 30 years medical work in Europe, Africa and in this country I am convinced that there is hardly any justification in making a diagnosis of pulmonary tuberculosis if, after careful and repeated examinations, tubercle bacilli are not found in the sputum of the patient, especially in the active stages of the disease. Even a clinically 'typical' case of chronic blood-spitting, cough with fever, progressive wasting and a significant family history thrown in, may turn out in the end to be no tuberculosis at all if the causative bacilli—the only infallible proof of the existence of the disease—remains constantly absent in the sputum. This statement is not intended to imply that it is impossible to diagnose pulmonary tuberculosis without the aid of a microscope. We know that phthisis was diagnosed with surprising ease and accuracy by the older clinicians when they had only their percussing fingers and wooden stethoscopes to depend upon! But it does mean that it is not always safe to diagnose pulmonary tuberculosis only on clinical findings. Under certain conditions even a X-ray picture may be exceedingly misleading.

It might interest the doctors serving in the present post-war army to know that error in diagnosis of pulmonary tuberculosis was committed on a huge scale during the last world war, when a large number of soldiers had to be hastily invalided out of the army as lung T. B. Everything went on smoothly for a time and then somebody at the Ministry of Pensions got suspicious, probably because the death rate among the consumptive pensioners was not high enough for all of them to be genuine consumptives, and suggested that a committee of experts should re-examine those cases. This was done and the result was astounding. Out of a total of 206 cases originally invalided as pulmonary tuberculosis, 87 at least certainly showed no signs of tuberculosis of any kind, at the subsequent examination by the experts—87 wrong diagnosis out of 206 "sputum negative" findings!

In this short contribution I wish to mention a few, not ordinarily looked for, hæmorrhagic bronchial conditions which might closely resemble pulmonary tuberculosis. As one who had the opportunity of being attached to a large military laboratory in the tropics during the last Great

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War I am in a position to state that one of the conditions which might easily be mistaken for pulmonary tuberculosis is "*broncho-spirochætos*" the offending organism of this trouble being a spirochæte, first discovered by CASTELLANI in Ceylon. In acute cases, these spirochætes are readily seen, often in large number, in "smear" preparations, stained by any recognised method or by dark ground illumination.

It will not be out of place here to say that broncho-spirochætos has always been more or less a controversial subject. Some observers finding it impossible to differentiate spirochæta-bronchialis from the harmless mouth spirochætes have discredited Castellani's discovery. One of them, TROCELLO, suggested that the mouth spirochætes could directly slip into the bronchi and appear in the sputum without significance. STRONG supporting NEUMANN and, to some extent, PONS, hinted the possibility of non-parasitic oral forms acquiring pathogenic properties by "mutation and adaptation" in the respiratory tract. In this connection it is noteworthy that an arsenical spirochæticide often clears up the condition in which the sputum reveals constant presence of spirochætes be it parasitic or saprophytic.

**Broncho-mycosis.**—Is another condition which simulates pulmonary tuberculosis. Mycotic infection of the respiratory tract is not rare, but it is only rarely diagnosed as such. The patients generally die as "consumptives" or a lung abscess may be the killing end. It must be remembered that a variety of fungi of different species and genera can grow in the human respiratory tube and set up symptoms more or less similar. In heavy infections the patient may present symptoms of phthisis with hectic fever and sanguinolent sputum. In lighter attacks there may not be anything more than a long-standing and troublesome cough which might disappear like any ordinary cough, under ordinary treatment. What is sometimes known as "tea-teasters" cough in Ceylon, according to ALDO CASTELLANI, is due to mycotic infection, often a broncho-moniliasis, as fungus monilia is fairly constantly present in the tea-dust. Here a word of caution seems necessary. An unimportant secondary broncho-moniliasis is sometimes seen along with pulmonary tuberculosis and other chronic chest affections. So the diagnosis of a purely primary broncho-moniliasis is by no means easy. Indeed, to decide whether any fungus seen in the sputum is important or not requires experience. Infections due to nocardia (broncho-nocardiasis) is said to resemble phthisis very closely and of all the mycotic infections of the air passage the most difficult to cure.

**Broncho-distomiasis.**—With cough pain in the chest, loss of flesh and hæmoptysis is not unlike pulmonary tuberculosis to the uninitiated. Fortunately the diagnosis of the condition is easy as the characteristic ova of distoma-ringri or paragonimus-wesermani, the offending lung fluke, is readily seen microscopically in the fresh sputum of the patient. Doctors from India now serving in the army operating against the Japs will do well to remember that this disease is met with in China, Japan, Formosa, Sumatra and other places.

**Conclusion.**—(1) Cough with bloody expectoration and emaciation does not necessarily mean consumption, more so in the tropics; (2) If microscopical examination of sputum remains constantly negative for T.B. suspect other possibilities and investigate further; (3) Remember that hæmorrhagic bronchitis of protozoal mycotic and helmentic origin may produce symptoms dangerously similar to pulmonary tuberculosis.

## Protein Shock Therapy—Theory and Application

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It is a method of treatment which is well designated non-specific, the term non-specific denoting a wide range of its applicability as well as the lesser degree of its specificity. Its action is dependent upon the fact that the introduction of foreign proteins into the blood stream causes a sharp reaction with a quickening of the metabolic processes, the reaction being a febrile one associated with rigors and sweating. The febrile reaction is followed by a degree of abatement of the symptoms—sometimes to an appreciable extent—usually in chronic diseases.

The foreign proteins may be introduced either subcutaneously or intramuscularly or intravenously. In using the former two routes one does not encounter the febrile phenomena. The action of proteins in these cases is dependent upon a degree of sensitization of the patients in allergic and sometimes in infectious diseases.

The substances used for protein shock are: (a) commercial peptone; (b) cow's milk boiled for 10 to 15 minutes in water bath; and (c) suspension of dead bacteria—vaccine—the most commonly used being T.A.B. vaccine.

**Technique for intravenous injection.**—Rest in bed, a light diet and continuous attendance by the medical attendant till symptoms of shock have passed off are essential. One cannot judge accurately the exact dosage of the proteins required for a particular patient. But to begin with one can give 20 c.c. of a 10 per cent. solution of Peptone or twenty-five millions of T.A.B. vaccine or B. coli vaccine suspended in a volume of 5 c.c. In case collapse ensues after the Protein administration it is best treated by Atropine Sulph 1/100 gr. or by Liquor Adrenalin Hydrochloride (1 in 1,000) 3 minims subcutaneously. The febrile phenomena come on within a period of one to six hours. Repeat doses are given at four-day interval, increasing them gradually. The scheme of dosage for T.A.B. vaccine are 25, 50, 100, 150, 200 and 250 millions of the organisms. The doses are required to be increased only when the reaction by the first and minimal dose has not been sufficiently severe.

**Technique for intramuscular and subcutaneous injections.**—While following these routes the patient is an ambulatory one, the scheme of dosage for Peptone being 0.5 c.c. of a 5 per cent. solution with a gradual increase in the volume by 0.2 c.c. upto a maximum of 2 c.c. The intervals maintained in between are of four days each.

**Application to diseases.**—1. *Rheumatoid arthritis*:—Its application in rheumatoid arthritis has achieved many good results but the successes attained are greater in the more recent cases; the more chronic the disease the less are the chances of success. The method of treatment though disagreeable to the patients often meets with remarkable lessening of the joint pains, freer movements and a good deal of general well-being. In addition to the substances mentioned above Colloid Sulphur (Crooke's) has been reported to be a very useful remedy. Given intramuscularly and beginning with an initial dose of 0.5 c.c., the doses are repeated at every 4th day and are gradually increased by 0.5 c.c. at each injection upto a maximum of 4 to 5 c.c. The reactions by use of this substance are milder and the

effects more lasting. When T.A.B. vaccine is used the joints should be passively moved and massaged within the period of reaction. In many cases though the results are encouraging they are apt to prove temporary. It should not be adopted in weak debilitated patients with a small degree of vitality.

2. *Hæmophilia*:—Its value in hæmophilia is restricted to infrequent checking of the spontaneous bleeding tendency into the joint cavities of the hæmophilics. Several things, most important of which is normal horse serum, are employed. Injections are given subcutaneously in doses varying from 10 to 20 c.c., repeated at monthly intervals. The doses in children are 2 to 4 c.c. of normal horse serum. The reactions are mild but the initial dose should not exceed the minimal amount possible. Other substances such as T. A. B. vaccine should not be used as they are followed by a degree of hypo-coagulability.

3. *Lymphogranuloma inguinale*:—It is of value if used while the glands are suppurating and if it is combined with aseptic aspiration of the suppurating glands. The injections are given at a three-day interval and continued till the buboes dry up and the induration in the surrounding tissues disappears.

4. *Anaesthetic leprosy*:—It merely supplements the treatment of leprosy by other methods by causing a healing up of the chronic leprotic ulcerations. But it is said to heal up early anaesthetic lesions very rapidly.

5. *Pemphigus*:—Protein shocks are best adapted to be given in this disease for the first four to six months of its onset. Later on no benefit is derived as the system is no longer able to call upon its reserves. T.A.B. vaccine is used intravenously with an initial dose not exceeding 5,000,000 organisms. But, anyway, it should not be used in Pemphigus Neonatorum.

6. *Tabes dorsalis*:—T.A.B. or B. Coli vaccine given in a suspension of Neosalvarsan intravenously are sometimes found effective.

7. *Undulant fever*:—It has got very good effect in cutting short the courses of Malta fever. In many instances only one shock suffices but generally four or more shocks are needed. T.A.B. vaccine in 30 to 50 million doses is employed.

8. *Obesity*:—It has been applied only in some refractory cases of endogenous obesity preliminary to thyroid medication with a view to render the tissues more susceptible to thyroid extracts. A preparation named Hypertherman (a suspension of B. Coli in milk) is considered to be the best for this purpose—given in a course one or two weeks prior to thyroid administration.

9. *Disseminated sclerosis*:—As no other treatment is known which may arrest the progress of the disease or cure, it is well to try this mode of treatment in some cases to induce artificial pyrexia. It may often be of benefit in some rapidly progressing cases.

10. *Epidemic cerebro-spinal meningitis*:—Certain anti-body preparations including the use of meningococci endoprotein have been used in the treatment of this malady but the results have not been satisfactory.

11. *Psoriasis*:—In view of the fact that every drug in Pharmacopœas has once enjoyed a vogue in the treatment of psoriasis and that there is

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no rationale of internal therapy in this disease, protein shock therapy has been given a chance. It has been said to have given beneficial results from time to time. It is worthwhile to give it a trial in obstinate cases.

12. *Raynaud's disease*:—In combination with other treatments for this disease such as thyroid or parathyroid administration, pancreatic extracts intramuscularly it may be employed for an early checking of ulceration or gangrene associated with this disease. Diathermy, Radiant Heat and Galvanism are its alternatives.

13. *Enteric fever*:—Although it was tried sometime ago in the treatment of this disease it has been completely given up now. It used to cause a rapid defervescence by lysis or even by crisis sometimes. Debility from other causes and hæmorrhages are its most definite contra-indications.

14. *Sycosis*:—T.A.B. vaccine given in small doses intravenously produces good and speedy results.

**Autohæmotherapy.**—No discussion on protein shock therapy can be complete without giving an account of auto-hæmotherapy. It is also a form of the same and works on the same principles. The patient's own blood is drawn from the cubital vein and is injected intramuscularly beginning with an initial dose of 5 c.c. and increasing it gradually to 10 c.c.—the increase being 1 c.c. at every injection. The course consists of six injections given at intervals of 6 to 7 days. In asthma, auto-hæmotherapy is used to sensitize the patient. The injection of 10 c.c. blood intragluteously sometimes causes temporary relief. It is also tried in resistant cases of carbuncle, 20 to 60 c.c. of the patient's own blood is injected all round the indurated area by making several punctures, the numbers of punctures depending upon the size of the indurated area and so also the quantity of blood injected.

#### Cases and Comments

### An Unusual Case of Fatal Coma

R. D. PENDSE, M.B., B.S.,  
Poona.

MEDICAL help was sought for a young boy, aged about 14, when one evening the boy became unconscious. Previous history was nothing particular and in no way illuminating. The boy had some loose motions that day and to all practical purposes the boy was not ill. It was in the latter part of the evening that he complained to the mother that he was unwell, and was feeling giddy. Soon he vomited, got fever and went to bed. He became unconscious within an hour or so by feeling giddy. When first seen the patient was completely unconscious. The temperature was 102°F. or so, the pulse was of good quality, full bounding and not fast though not slow. But the most striking feature was the irregular breathing. The period of breath-holding or apnæ was quite noticeable and one would think that the patient was dead. But within a fraction of a minute or so the breathing would start, with a jerk at the cervical muscles on the right side. This localized convulsive movement was restricted to the right side of the neck only. There were no generalized convulsions either at the beginning or during the entire course. Once the breathing started there were no longer any jerky movements, the breathing continuing sometime then again stopping once more. The

breathing resembled to a great extent that described as Cheyne Stokes. There was no cyanosis even during the period of apnæia. The eyes were not congested, the pupils were somewhat contracted, not reacting to light and the corneal reflex was not present. There was no nystagmus, nor any deviation to any particular side. Other reflexes could not be elicited, but there might have been some Kernig but it was not definite. There was no rigidity of neck. The limbs were neither flaccid, nor rigid nor paralyzed, their tone being normal. There was no œdema on the body nor any coldness of extremities, nor any sweating. There was no history of any injury nor any likelihood of poisoning. There was no obvious smell to the breath. Heart and lungs were normal in the aging but at the end which came within about two hours or so, the heart began to fail, and the lungs showed acute œdema. Urine could not be examined and the throat was not examined as there was no history pointing to its involvement.

Next lumbar puncture was done. The fluid was slightly under tension but was quite clear. Next morning there was no clot formation—cow-web or any turbidity. No further examination of the fluid was done as the patient was dead some hours. A blood film had been taken, and it was examined next morning out of curiosity but no M.P. could be found.

The patient was given intravenous Glucose 25 per cent. with Quinine and Coramine, but the patient soon died of acute pulmonary œdema within about four hours of onset.

**Discussions.**—For adopting treatment it was necessary to arrive at a reasonable diagnosis. First of all there were two common possibilities, the first being cerebro-spinal meningitis, and the second one still commoner possibility of cerebral malaria. The result of lumbar puncture was that there was free clear fluid, almost under normal tension. There were two prominent symptoms in the case, the first being rapidly developing coma, and Cheyne-Stokes breathing. The third feature though not so marked was the moderate fever. It is unnecessary to go over all the causes of coma, such as acute fever, uræmia, diabetes, intracranial lesions, exogenous causes like alcohol, opium, etc. The conditions where Cheyne-Stokes breathing is to be found are: (1) acute fevers; (2) raised intracranial pressure; (3) condition of heart failure; (4) uræmia; (5) opium poisoning; (6) any cause of anoxæmia; (7) conditions associated with degeneration of cerebral arteries.

Cheyne-Stokes breathing occurs amongst infants and sometimes among the aged where this occurrence may be physiological. The exact causation of Cheyne-Stokes breathing is not well understood, the general explanation being anoxæmia of respiratory center or oxygen lack. This satisfactorily explains its association clinically with pneumonia, congestive heart failure, opium poisoning-conditions which clinically are conspicuous by failure of satisfactory oxygenation of blood. It is less easy to understand why it also occurs in conditions associated with raised intra-cranial pressure. Clinically Cheyne-Stokes breathing has not much diagnostic significance but it is more important from the point of prognosis, the outlook generally being gloomy in conditions where it is present. It is mentioned that in cases of raised intracranial pressure the cycles of rapid breathing are of short duration and the regularity with which waxing and waning occurs is also not so well marked. There are sub varieties of Cheyne-Stokes breathings that are described but prime distinguishing characteristic is the same,

the period of breath holding intervening with waxing and waning respiratory cycle.

**Differential diagnosis.**—1. *Cerebro-spinal meningitis*:—The first consideration was to exclude or establish this diagnosis. Absence of rigidity of neck, Kernig sign, and above all clear C.S.F. under normal tension, exclude this possibility. However it is said that fluid may be clear during the first twenty-four hours or so of the onset of infection. The blood film examined next day did not reveal polynuclear leucocytosis. Again it is possible that the fluid may be clear in fulminating type of cases where the body is so overpowered that the defence mechanism does not come into play. However in this case in spite of coma the general condition of the patient and the quality of the pulse which was quite good does not fit a overwhelming infection. In that case the general condition would have been very poor, temperature much lower and the pulse flickering. In C. S. meningitis death would not come so early from the onset. Cheyne-Stokes breathing may occur in meningitis as it may occur in any intracranial involvement, but then this is a late phenomena, indicating great pressure and asphyxiated condition of the respiratory centre. Cheyne-Stokes breathing is then an expression of raised intracranial pressure, and other corroborative signs such as kernig, rigidity of neck, contracted pupils, convulsions would not be absent. Raised intracranial pressure would exert its adverse effects on all structures in the cranium decidedly more or less on different structures but it is hard to accept that the pressure would only be exerted on the respiratory centre.

2. *Cerebral malaria*:—This appeared the next possibility specially when the C. S. fluid was found clear and under normal tension. Quinine was administered as it was thought undesirable to wait till the result of the blood examination would be available. Blood film was made then but it was examined next day after the death of the patient. It did not show any parasites. Quinine was given intravenously.

3. *Epidemic encephalitis*:—Rapid coma with fever is not common. The acute form is very rare, and the symptoms are so slight that the acute stage is generally missed. But the onset may be very acute and abrupt, characterised by high fever, delirium and convulsions. In its more usual form nervous symptoms make their appearance insidiously such as, ocular palsies, 7th nerve paralysis, difficulty in swallowing, and speech. The C. S. F. does not show any characteristic changes and appears normal. However there were no cranial nerve palsies. The respiratory rhythm may be disturbed in this affection, the disturbance being of Cheyne-Stokes type or breath holding attacks in which the apnæic stage may terminate in convulsion. In this case there was no general convulsion at the end of the apnæic phase but a convulsive jerk at the cervical muscles. There were no generalised convulsions nor cranial nerve involvement and it would appear that the intra-cranial affection was strictly limited and not diffuse as it would be in encephalitis.

4. *Acute poliomyelitis*:—The usual form is spinal with lower motor paralysis of limb muscles. Though the virus has a liking to affect the spinal grey matter, there are reported cases of the virus affecting other parts of the central nervous system, like the cerebrum, mid-brain cerebellum etc. With the involvement of the cerebrum there are convulsions, twitching and spasm of muscles, and muscular rigidity. Cerebellar involvement produces

acute cerebellar ataxy. The involvement of the brain stem produces various cranial nerve palsies, 7th being common, paralysis of soft palate, with regurgitation of fluid, and a nasal voice are produced. C.S. fluid is clear and on standing a fine coagulum may form. The virus of poliomyelitis may affect very localized areas in the nervous system, producing palsies of solitary muscles. It may appear that in this case it might have happened that it only affected that portion of the medulla oblongata where the respiratory centre is situated, giving rise clinically to Cheyne Stokes breathing and rapid end. Death was caused not by the virus infection itself but the infection settling on a very vital structure—the respiratory centre, failure of which was mainly responsible for the quick termination. This is merely a conjecture, and the writer even now after consulting books and medical friends, is not satisfied with the above conjecture. Why the respiratory centre alone without the neighbouring structures being least affected should have borne the brunt of infection is not clear.

5. *Other forms of meningitis*:—It is unnecessary here to refer to septic meningitis and that caused by pneumococci, because in these the C. S. fluid is not clear. In tubercular form the fluid is clear but the whole picture is different, extending over some days. The C. S. fluid on standing shows quite often the characteristic cobweb clot. In benign lymphocytic and serous meningitis the fluid is clear, but in both the course is never so rapid, and the termination is usually in recovery. Mention may be made here of syphilitic meningitis, not because it is an acute condition, but sometimes it starts not in that common chronic fashion but has a dramatic onset. The fluid is clear and it may be positive to Wassermann, the blood being almost so.

**Treatment.**—In cases where diagnosis is not possible the only thing that can be done is to keep the strength of the patient, and try to remove any adverse factors that may be operating. Oxygen therapy might have been used. Aminophylline is said to abolish Cheyne-Stokes breathing, but whether it succeeds only in cardio-vascular cases or whether it is useful in cases of intracranial lesions is not definitely known. Oxygen therapy is said to be more efficacious in cases of intracranial origin. Glucose in higher concentration, 50 per cent. or so, may be useful in relieving raised intracranial pressure. Mag Sulph injections may serve the same object. Urea is now becoming more popular for relieving pressure headaches.

Regarding the treatment given to this patient, specially administration of Quinine intravenously, may evoke some criticism. It should be the general rule to establish the diagnosis before specific drugs and specially potent drugs are used. But in private practice where all laboratory facilities are not at hand, and in cases of emergencies drastic measures have to be taken. It was impossible in this case to wait till the blood could be examined, because the condition of the patient, was so desperate, and he was going down so rapidly that it appeared the only chance to take was that the case may be of cerebral malaria. Giving Quinine is decidedly less dangerous than to allow the case to slip by mere watching and to find when it is too late that it was cerebral malaria. Cerebral malaria is not an uncommon occurrence and it should always be considered in all cases of fever and coma.

## A Case of Abnormal Permeability of Retinal Vessels

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Rama, male, 30 years, married, history of enteric fever in his twelfth year. No other illness till now of worth mentioning.

Complaint: Blurring of vision in the right eye.

**Present history:**—In the middle of January, '45, when the patient got up from his bed early in the morning, he noticed that his vision in the right eye was hazy. There was a thick cloud—more denser at some places as to obscure his vision when he attempted to read or write and thinner at some points as to allow him to see things perfectly clear—in the field of vision. Scattered throughout the cloud, rather closely, he could see numerous transparent round bodies floating. Both the cloudy opacity and the round opacities move with the movement of the eye and not fixed in the field of vision. He felt them all floating before his eye. Within 8 to ten days the cloudy opacity slowly disappeared but not completely leaving a few streaks of transparent opacities which continued for a long time but did not obstruct vision. Particularly, there was a U-shaped transparent opacity, in the centre of the field of vision, which appeared as thick and black, surrounded by the round opacities at the beginning of every attack. This U-shaped opacity also slowly disappeared but not completely and is still present. The transparent round bodies became thinner, turned into black dots and finally disappeared completely. Patient used to see clear and beautiful hollows around the lights during nights.

The next attack was in the middle of February '45 when it was diagnosed as retinal hæmorrhage and treated. The attack repeated almost every month, occasionally twice in the same month, and the absorption of the opacities was as quick and complete as in the initial attack. The attacks used to commence when he woke up from bed early in the morning except on two occasions, once in the afternoon after two hours sleep and at another time in the middle of the night. There was no relation to hard physical exercise he used to take. There was no pain at all at any time. It always occurred in the same right eye up to now. The intensity of the attack increased from morning till the afternoon and then slowly it subsided. He had to move his eyes continuously in order to avoid a denser opacity to read or to write. Even on the first day of the attack he was able to read 6/6. When he closed both his eyes and turned towards a bright light the red glow in the right eye was more intense and opacities more clearly seen than in the left eye. There were eight attacks till now.

**On examination:**—A well nourished patient, takes to active sports. Not anæmic. All systems of the body nil abnormal. Both eyes equal in size and shape. No squint. No nystagmus. All the movements of both eyes normal. Conjunctiva and cornea normal. Pupil equal on both sides, react to both light and accommodation. Anterior chamber normal on both sides. Lachrymal passages patent on both sides. Distant and near visions normal. Ophthalmoscopically: Lens normal. Vitreous hazy with white streaks at the beginning of the attack, later the haziness disappeared but only streaks of opacity seen, particularly the U-shaped opacity. optic disc and retina normal. Intraocular tension and fields of vision normal.

**Clinical findings:**—Urine: Nil abnormal; Blood Pressure: 115/75 mmHg; Blood clotting time: 3.5 minutes; Blood calcium: 11.5 mgms.; Thrombocytes: 130,000/c.m.m.; Night blood; No microfilaria; Blood film: Normal; Hæmoglobin: 90%; Blood Wassermann and Khan reaction: Negative.

**Treatment adopted:**—(1) Complete rest during attacks and a brisk purge either with castor oil or Epsom salt; (2) Advised to wear Crook's dark glasses; (3) Calcium by mouth and by injection; (4) Iodides by mouth; (5) Tonics like B.G. Phos. by mouth; (6) Dionine eye drops; and (7) Vitamin C<sub>2</sub> by mouth and by injection.

In spite of this treatment for considerable time, the attacks continued. The patient was advised to take regular vigorous physical exercises early in the morning, and he was doing since three months. He was asked to take life easy and nothing was given in the form of medicines. Now he had no attacks since three months, and his physical condition is excellent but the patient says streaks of transparent opacities still persist. He was advised not to worry about them and was assured that the opacities would get absorbed in course of time. But still that U-shaped linear opacity continues.

**Points of interest:**—1. Attacks in the same eye almost every month with the same intensity.

2. All opacities are floating in nature and take 8 to 10 days to disappear.

3. There is a particular U-shaped opacity in the centre of the field of vision, which never disappeared completely, neither it changed its shape or place. This floats more with upward and downward movement of the eye than sideward movement. This might have formed below the rounded body of vitreous and somehow escaped to the centre and is anchored there though floating.

4. The attack begins either in sleep or early in the morning when the patient is at complete rest.

5. All systems of the body are normal except for the slight thrombocytopenia which may be the cause for the capillary permeability.

6. There is absolutely no pain at any time.

7. Both eyes are absolutely normal in all other respects and the patient does not wear glasses at all.

**Discussion.**—In the above picture, there is nothing which gives an irrefutable diagnosis of irido-cyclitis, retinitis or retino-choroiditis or tuberculosis of uveal tract. There is nothing to suggest active hæmorrhage into the vitreous either from the iris, choroid or retina. It is not even intra-retinal or sub-hyloid (pre-retinal) hæmorrhage. It does not fit in Eales' disease though some points are in favour of it. It looks as though there is some abnormal capillary permeability from choroid or retina particularly when the patient is at complete rest, the albuminous fluid with the corpuscles diffusing into the body of vitreous. The slight thrombocytopenia may be the cause for the increased capillary permeability.

But why it occurs only in one eye, and even that at periodical intervals when the body is at rest, is not to be explained. I leave it to my medical colleagues for further comments and suggestions.

I offer my grateful thanks to Dr. Janab Mahomed Ismail Saheb, M.B., B.S., D.T.M., District Medical Officer, Nellore for his kind suggestions and for allowing me to publish this case.

## A Bad Case of Status Epilepticus

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THE following case report might be of interest to my moffussil fellow brothers in view of the fact that sometimes they find it very troublesome to treat a bad case like epilepsy. This is generally resistant to any treatment.

On 12th December 1945, I was called in to attend a Hindu girl, Gouri by name, aged 9 years, suffering from fits which followed one another at short intervals for the last 2 days.

**Past history.**—The girl was of good physique and healthy with no other ailments up to 3 years of her birth. She was the 1st issue of her healthy parents in normal delivery and in full term. No history of syphilis of her parents. When she was  $3\frac{1}{2}$  years old, she had an attack of high fever. For 15 days during which period water was not applied on her head at the high rise of temperature in adequate quantity, temperature every day rose up to  $104-105^{\circ}\text{F.}$  during noon and came to normal in the evening with high rise again in night which came down to normal in morning again. No convulsion was noticed during high rise of temperature though the patient abnormally cried and face and eyes became blood-shot due to rise of blood in brain.

After 2 injections of Quinine Bi Hydrochloride and about 40-50 grains of Quinine by mouth temperature subsided after 15 days. After 15 days of the fever when the girl was quite O. K., suddenly one noon, she fainted in her father's lap and convulsion took place for 2 minutes. After cold drainage over her scalp, convulsion subsided and the girl fell deep asleep. After sleep she was quite alright and could not remember that anything happened to her. The parents after this thought the convulsion was due to worms and gave her medicines for worms. After 20 days the girl who was hale and hearty, while standing by her mother, suddenly dropped down backwards and similarly convulsions began and foam came out from her mouth and the girl lost her consciousness. After few minutes the girl fell deep asleep. After that similar fits occurred after interval of 15 or 20 days but at night and during sleep. The girl was then brought to Calcutta and examined by eminent physicians who diagnosed it to be a case of epilepsy and prescribed Luminal gr.  $\frac{1}{2}$  morning and evening and Peacock's Bromide 3 p. at noon and bed time. The girl continued this prescription for 3 years with no good result. Fits after 20 or 25 days occurred at night time once or twice and at day time the girl was very irritant and unruly, beating her sisters and brothers and eating very much. Though the girl had so many fits no mental defects were noticed in her. She was as sharp as before. Before fits girl became puffy and after fit she again became reduced.

After 3 years the girl regularly took Dilantin Sodium Capsule P. D. gr.  $1\frac{1}{2}$  thrice daily for one year without any marked effect. Again Luminal was given to her regularly. On the last November as the parents could not procure Luminal, practically no medicines were taken by the girl and as a result fits became very frequent. On the 12th December fits followed one another at short intervals but with no complete loss of consciousness.

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I examined the fit, there was tonic spasm, upper extremities flexed, lower extremities extended, body arched backwards and with conjugate deviation of eyes. The larynx got into spasm, the respiration stopped, and there was cyanosis. Next relaxation followed—there was a period of clonic movements, foaming at the mouth. Urine passed during the fit. As soon as one fit subsided again another fit developed. Loss of consciousness was not complete.

**Heart:** Both sounds are feeble and rapid; pulse: 110 p.m.; respiration: 26 p.m.; lungs: free from any abnormality; eyes: congested, pupils widely dilated on both sides, conjunctival reflex—partially lost; tongue: marks of biting were seen with extreme dryness; bowels: constipated; temperature:  $101^{\circ}\text{F.}$ ; spleen: palpable 2 fingers below costal margin as the patient had an attack of malaria a fortnight back and got cured after 2 injections of Quinine 10 grains and 20-30 grs. of Quinine by mouth. Reflexes were found lost with extensor plantar reflex.

From the past history and clinical findings of the patient I diagnosed the case to be status epilepticus with grave prognosis.

**Treatment.**—I gave her glycerine enema as the patient had no bowel for last 2 days. No action with enema though the patient tried to pass stool. Then I introduced by left index finger with application of Glycerine on it into the rectum and extracted with great difficulty hard faeces just like stone in profuse amount. After the hard faeces were extracted the patient passed a semi-liquid motion herself with offensive decomposed smell. As Sodium Luminal or any other like medicines were not available to stop the seizure of continuous fit I injected at once gr.  $\frac{1}{4}$  of Morphia and waited by her side for 3 hours. But the patient did not sleep even with  $\frac{1}{4}$  gr. of Morphia and fits continued one after another. Taking courage in both hands I again pushed further  $\frac{1}{4}$  gr. of Morphia with  $\frac{1}{100}$  gr. of Atropine. But no improvement was noticed. Cold application of water was always continued. I then really got nervous and called Dr. KUNJA LAL MAZUMDAR, B.Sc., M.B., of Gunaigacha. He came in the evening and examined the patient and was astonished to see that even 2 injections of Morphia gr.  $\frac{1}{4}$  each could produce no sleep. He then prescribed Peacock's Bromide 3 p. every 3 hours. Even after 3 or 4 doses of Peacock's Bromide no improvement was noticed and the patient had not a single minute sleep and fits similarly continued. Next day Dr. MAZUMDAR was called again for consultation. He then injected another gr.  $\frac{1}{4}$  of Morphia and Peacock's Bromide was continued. But despite so many Bromides and 3 injections of Morphia no improvement was noticed and the patient gradually went down hill. On the 6th day I noticed that during the fits the patient told that she was being frightened. Thinking it to be a case of hysteria I prescribed Elixir Bromovalerianate (Sabail) one dram every 4 hours alternated with Peacock's Bromide  $\frac{3}{4}$  i. But no improvement noticed, and the patient could not take any diet for so many days. Acidosis was noticed and the patient became very weak for want of nutrition. Only a little quantity of Glucose D water was given to the patient forcibly when the fit subsided. No temperature was noticed on the 5th day though there was spleen. Quinine injection was not intentionally given to her with the fear that it might irritate and increase the fit. On the 8th day I gave all hopes about life of the patient and injected one ampoule of Cardiazol to prevent immediate heart failure which I expected every minute and put constant application of ice over the head for the first time. To my utter surprise the patient who had no

single wink of sleep for 8 days despite Morphia injections, Bromides—Bromovalerianate, etc., suddenly fell into deep sleep. I thanked God and passed the night with patient, and constant application of ice was continued. The patient had a sound sleep the whole night and fits subsided. Next morning when she woke up she complained of severe headache and extreme hunger. I found the condition of her heart much better. I gave Peacock's Bromide 3 i every 4 hours and gave gruel for her diet. Though continuous fit subsided petitmal continued and the patient was saved this time. She is now having petitmal everyday and Peacock's Bromide still continued. Coramine 1 c.c. was injected for 3 days (Ciba), as the patient's heart was very weak.

I shall feel highly obliged if any of my fellow brothers and the learned Editor of this widely circulated and much popular Monthly Medical Journal 'ANTISEPTIC' discuss the case note and kindly throw a light about the treatment of the case—diagnosis of the case and future care and diet of the patient so that her life may be saved. This time she looks morosed and her face has become dull and she loses her memory suddenly for a minute or two with regaining of memory again, i.e., slight mental deterioration is noticed after the status epilepticus.

### Importance of Primary Disinfection in Wounds Caused by Dog-Bite

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It is an admitted fact that once rabies makes its appearance no hope of recovery exists. But thanks to Pasteur's efforts much can be done to prevent its establishment by producing immunity. The process of production of immunity is a long one and the incubation period may in some cases be as short as 10 days, so anything which can prolong the incubation period is of great importance. Cauterization with fuming nitric acid within five minutes of the occurrence of a rabies wound prevents development of the disease; within  $\frac{1}{2}$  hour of infection it prevents a certain proportion of cases from developing. Subsequent cauterization prolongs the incubation period which is helpful in establishing immunity. To illustrate the effects of cauterization I cite below the few cases I have come across in my practice.

**Case 1.**—35 years m. attended the rural dispensary, Indri, on 31-1-39 within an hour of infliction of wounds. They were dog-bite wounds on both of his hands and forearms. Wounds of the right side were more in number and more lacerated. I cauterized them with fuming Nitric acid and dressed aseptically. Being afraid of the pain caused by cauterization he assured me that the dog was a pet one and it was not rabid. So I washed his left side wounds with Condy's lotion, applied Liq. Iod mitis and dressed aseptically. On the fourth day the dog became rabid and the man at once went to the nearest clinic where anti-rabic treatment was given for 14 days. His wounds healed uneventfully within a week. On 8-2-'39 he felt some pain along his left arm and shoulder for which he was given Sod. Salicylas mixture internally and lint. Terebinth for massage. Next day he got some headache, malaise and slight fever. His healed wounds on the left side became

tender and painful. On the 4th day he felt inability to swallow the liquids, I was summoned to see him. He complained of intense thirst and insomnia and his voice was hoarse. He was maniac and restless and had high temperature with very rapid pulse. Passing through the excitement and paralytic stages the poor fellow died on the 6th day of illness. It was noted that no pain was felt on the right side.

**Case 2.**—A 14 year old sweeper girl was brought to me from a neighbouring village on 1-3-'39 for inability to swallow, insomnia and excitement. Thick saliva was dribbling out of her mouth. She was bitten by a she-jackal on her left leg 3 months previously and the wounds were dressed with red Capsicum.

On 22-3-'44 a rabid dog bit 3 men, 2 she camels and 2 dogs. All the animals died of rabies. All the 3 men attended my dispensary (Nos. 3, 4, 5.)

Case 3 and 4 reached the dispensary within 15 minutes of infection of wounds. There were teethmarks on both extremities. All wounds were cauterized with fuming Nitric acid and dressed. Wounds healed in a week. They took no anti-rabic injections. They have not developed the disease upto 1-1-'46.

**Case 5.**—18 years m., reached the dispensary within 20 minutes of infliction of wounds. He had many extensive and deep lacerated wounds on both his limbs and face, some of them were bleeding profusely. Thorough cauterization with fuming Nitric acid was done and aseptic dressing applied. He too did not go for prophylactic anti-rabic treatment. In a fortnight his wounds healed completely and he resumed his work. In the middle of June '44 he felt weakness of his lower limbs and some difficulty in micturition. He developed pain followed by paralysis of legs and incontinence of urine, became maniac and could not swallow water. He was taken to another village where he died. All this took only a week's time. I had no opportunity to see him in his illness.

**Comments.**—From case No. 1 it is plausible that although wounds of the right side were more extensive yet due to cauterization infection did not spread from there. The incubation period was short and the prophylactic injections could not prevent the establishment of the disease. The totally untreated case No. 2 developed symptoms in 3 months' time. Cases Nos. 3 and 4 did not develop the disease although no anti-rabic injections were given and only early cauterization was done. Case No. 5 is probably an instance where cauterization prolonged the incubation period; otherwise he would have developed the disease much earlier as he had numerous extensive wounds of upper extremities and face also. Case No. 5 could have probably been saved by prophylactic injections.

**Conclusions:**—1. Early cauterization of a dog-bite wound is very essential.

2. Rabies may occur after full course of prophylactic injections. The nearer to the central nervous system the wound is the shorter is the incubation period as the virus travels centripetally.

**Acknowledgement.**—I am thankful to Dr. Karnal for his permission to publish this note.

## An Unusual Case of Diabetes Mellitus

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I WAS recently called to see a Hindu male child of about 5 years of age with the following complaints:—(1) Polyuria with severe thirst; (2) Extreme emaciation.

I saw him in the following condition:—

- (1) Extremely anæmic and emaciated.
- (2) Frequent micturition—even thrice in an hour, the quantity each time being 4 to 6 oz.
- (3) Voracious appetite and severe thirst—a glass of water was always kept near him.
- (4) Oedema of extremities and slight ascites.
- (5) Tem. 98°F. P/R—120/98 per minute.
- (6) Spleen not palpable—Liver greatly enlarged.
- (7) Clear tongue—chronic constipation.

The following history was elicited:—

About six months back the boy got an acute attack of malaria with temp. of 105°F. Quinine Bihydrochlor gr. v in 1 c.c. was injected in the gluteal region. Next day, the temperature not remitting, another Quinine injection was given in the same place (through mistake.) The part became greatly inflamed and it was reported that one doctor opined that pus had formed and the part needed incision. But the operation was not agreed, neither the abscess burst by itself. The inflammation remained very great for about 15 days and then gradually began to subside and the part became normal in another 15 days. A few days after subsidence of the inflammation the boy began to pass urine more frequently than usual. He was under Homeopathic and Kabiraj treatment for about 5 months but his condition became worse day by day till he was in the present very bad state. The parents of the boy are living and healthy of only 30 and 22 years of age. There is no family history of diabetes in the paternal or maternal side.

The urine of the boy was tested by Fehling solution—on the slightest heating, the solution became deep red in colour. Specific gravity was 1028.

As it was impossible to give modern treatment of diabetes under the unfavourable circumstances of his house, removal to hospital was advised which was refused.

It was clear that the acute infection by destroying the islets of Langerhans caused the glycosuria. This report is sent because an infection causing diabetes in a child of 5 years is extremely rare and is surely interesting.



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MANUFACTURED BY:—THE ARMOUR LABORATORIES, LONDON.

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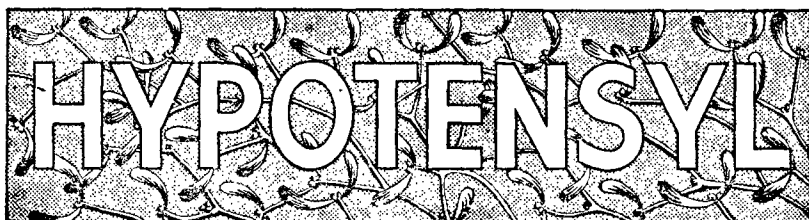
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### Rehabilitation

The War that has just ended has been largely responsible for the development of Rehabilitation. Uptil now, Medical and Surgical treatment meant no more than the cure of disease and the repair of injury. But this war has shown us the necessity of speedy return to duty of the sick and wounded soldiers without loss of physical fitness and also the importance of preventing unnecessary invalidism or disability amongst essential war workers. Governments of various countries in the world have recognised the importance of this aspect of social medicine and have drawn up plans for doing research in this line and for opening up more centres for this purpose. We hope the Government of India will fall in line with other civilised Governments and take up this question enthusiastically.

Long illness and severe injury have been known to produce prolonged immobilisation, and this in turn results in a condition of physical deterioration which can only be tackled by active physical means. As a result of this prolonged immobilisation muscles become atrophied, dependent soft part oedematous, injured soft tissues become adhesive, and joints become stiff. It is only by means of active exercises that circulation is properly maintained and the ill-effects of prolonged immobilisation prevented, or rectified. This, which was first recognised in the treatment of fractures, has now been applied to other injuries as well. Injuries of soft parts, extensive burns and infection of deep structures all respond well to early movement, accompanied by appropriate physio-therapeutic measures and suitable occupational therapy. In England this principle has been applied extensively to the restoration of vital capacity after serious affections of the chest. Patients who have undergone such serious thoracic operations as lobectomy, pneumonectomy, and thoracoplasty go through a special course of breathing exercises before and after operation. Similar methods are employed in the treatment of pneumonia, empyema, emphysema and other conditions where there is respiratory defect or lack of chest expansion. These principles have also been utilised in the treatment of cardiac disorders. Experiences at Service Hospitals during the present war has clearly proved that post-operative chest complications and recurrent hernia have been very much reduced by the adoption of such carefully planned course of breathing and abdominal exercises both before and after

abdominal operation. It is claimed also that the risk of thrombosis and of visceral prolapse is considerably reduced by the adoption of these principles in child birth. It is also presumed that gastric and intestinal disorders involving long periods of rest in bed, would also react favourably by such well-planned programmes, exercises, and occupational therapy.

In the field of neurology, this treatment has also been found very useful. Functional disorders, hysteria, effort syndrome, anxiety neurosis, and other types of disturbed mental equilibrium are treated today, not only by psychiatric investigation, narcosis, insulin and electric shock therapy, but also by a carefully balanced programme of physical exercise and occupational therapy.

The heavy incidence of peripheral nerve injuries in the present war has opened a wide field for the institution of these methods. During the long period between the suturing operations and the recovery of motor and sensory function, weak or paralysed muscles supplied by these nerves, have got to be maintained in a proper tone and strength. Their circulation must be kept up, contractures prevented and co-ordination maintained. In these cases, hydrotherapy, massage, electrical stimulation, finger exercises and selected occupational therapy, all play an important part in restoring physical function.

In the field of plastic surgery also, rehabilitation has advanced enormously. Here also the combination of physiotherapy exercises contribute to the production of healthy, supple grafts, mobile joints and helps to remove the depression from which such patients usually suffer. The above illustration does not by any means exhaust the examples where rehabilitation has been successful. The re-education of patients who have lost their limbs, the treatment of the blind and the deaf in preparing them for useful employment in later life, the combination of graduated exercises and sheltered employment in the tuberculosis sanatorium or colony, the special clinics and hospitals for the treatment of children with congenital deformities and other forms of orthopaedic disability, all represent conditions to which appropriate forms of medical or surgical rehabilitation are applicable, and without which treatment can never be complete.

The basic principles which govern the technique employed in the restoration of physical and psychological function, after serious illness or injury, are fundamentally the same whatever the nature of the disability. They involve the combined use of physiotherapy, physical exercise alternating with periods of complete relaxation, occupational therapy and handicrafts, games, psychological adjustment and vocational guidance. In carrying out this combined programme, the services of a team of trained workers cannot be over-emphasized and success and good results have been achieved where such trained men work together. For details with reference to the technique of rehabilitation in its various stages and the place where such stages must take place are too much of a detail which cannot be considered in this small article. We would refer our readers to the very excellent article on the subject by HAROLD BALME, O.B.E., M.D., F.R.C.S., D.P.H., Medical Officer-in charge of Rehabilitation, Ministry of Health, who has written a very beautiful article in the November 1945 issue of our distinguished contemporary *The Practitioner*.

The main purpose of rehabilitation is to see a man through from the hospital ward to a suitable occupation in life and this necessitates the

closest possible liaison between hospital, employment exchanges and industry.

It is not the part of the State alone but large Industrial Companies and Industry in general, must soon realise that the care of the individual worker, his protection from physical break-down and accident, the provision of best possible specialists' care for the worker, the encouragement of measures for his physical and mental rehabilitation, the financial care of his family during his period of illness, incapacity and re-training and the institution of appropriate means of his resettlement are matters of joint concern and a matter of vital importance to future national economy. It is upto the governments of the future to see that this view is recognised and to do their best.

## Gleanings From Medical Press

### MEDICINE and THERAPEUTICS

**Hepato renal syndrome.**—The name is confined to conditions in which primary hepatic disease is followed by signs of renal failure. The syndrome in its classical form is seen after operations on the gall bladder or biliary tract and after mechanical injuries of the liver. It may, however, occur with any severe hepatitis or necrosis of the liver. It is not generally appreciated that degenerative changes of varying degree are always found in the kidneys of patients dying from acute infective hepatitis and homologous serum jaundice. The lesions range from simple cloudy swelling to actual necrosis.

There are two schools of thought about the mechanism of production of renal damage. It is similar to the renal damage which results from crush syndrome, gas gangrene or incompatible blood transfusion. One school ascribes it to the action of toxic substances, such as products of digestion, which are detoxicated in the normal liver, products of liver autolysis, and the toxins of the micro-organisms in the liver or the biliary passages. The other school ascribes the renal damage not only in the hepato-renal syndrome but in all these disorders, to vascular shock, dehydration, and failure of the blood supply to the kidney.

The primary objectives of therapy in cholaemia and the hepato-renal syndrome are to repair dehydration and restore the disturbed blood chemistry to normal, to reduce the metabolic load on the liver and kidneys and to promote the regeneration of cells which have been destroyed. It is desirable to have frequent estimations of the blood urea, blood sugar, plasma chloride, and plasma bicarbonate, and a chart should be kept of the fluid balance. If parenteral therapy is used, the greatest care must be taken to avoid circulatory overloading, oedema of the lungs or abrupt alterations in the blood chemistry. Glucose should be given freely, by mouth or intrave-

nously. Calcium salts are of value for the relief of symptoms of cerebral irritation. Barbiturates are dangerous and should not be used for intravenous anaesthesia. If there are signs of vascular collapse, transfusion of plasma is probably desirable. Orthodox therapy is to restrict the intake of both proteins and fats to reduce the work of the liver. Recently there has been a tendency to give a high protein intake, in the belief that proteins may protect the liver from fatty infiltration and necrosis and promote the regeneration of the parenchyma. Data are still inconclusive. There is a stronger case for choline chloride, as it is unlikely to do harm if injected slowly, and it is believed to influence the kidneys as well as the liver. A daily dosage of 10 g. has been recommended, and this should be given by slow intravenous drip in normal saline not exceeding 3 g. choline an hour.—*B.M.J.*

**Salicylate or Sulphonamides.**—That sulphonamide therapy is not only useless in acute polyarticular rheumatism but may have most disastrous results is shown by E. Jequier-Doge, (*Revue Medicale de la Suisse Romande*, September 25, 1945, 65, 542). Among illustrative cases recorded is that of a man of thirty-six suffering from a recurrence of acute polyarticular rheumatism following an attack of furunculosis with impetigo. Sulphonamide therapy failed completely and a series of complications in the form of broncho-pneumonia, cerebral and pulmonary emboli, glomerulo-nephritis, petechiae, buccal ulceration and further furuncles developed. The treatment was changed and salicylate in daily dosage of 6 grammes substituted, with resultant stabilization of the patient's condition, although fever still persisted. When the dose of salicylate was raised to 10 grammes daily the result was dramatic; the temperature returned to normal in three to four days, and on discharge the patient was cured,

apart from a compensated mitral insufficiency. Not only is salicylate specific for the treatment of acute polyarticular rheumatism but its administration is of value as a diagnostic test, as the prompt response to the drug serves to differentiate between this condition and sepsis. In spite of signs of intolerance the sodium salicylate should be given in sufficiently high initial dosage; 8 to 10 grammes daily is the minimum dosage recommended for an adult, which can be instituted by the administration of 6 grammes on the first day, rising by 1 to 2 grammes daily to 12 grammes per day. The irritant effects of the drug can be prevented by division of the dose, by giving the drug in solution form, by giving the solution with a little milk or bicarbonate, or in gelatin capsules, or by giving part of the daily dose by intravenous injection in solution of 5 or 10 per cent. The rectal route can also be employed. The dosage should not be progressively lowered until forty-eight hours after signs of amelioration, and a maintenance dose of 3 or 4 grammes daily should be continued for some weeks or months. Signs of intoxication are unusual but should they occur, in the form of eruptions with febrile reaction, delirium, toxic dyspnoea and cyanosis, the treatment must be stopped immediately.—*Practitioner*.

**Mechanism of diabetic coma.**—It cannot be said that diabetic coma and hypoglycemic coma are both the result of failure of the brain to burn glucose. For, while this is true in the latter condition, because of a failure of delivery of adequate amounts of glucose to the brain as a result of the low blood sugar levels, it has been shown that in the total absence of insulin the brain is able to metabolize carbohydrates and maintain its respiratory quotient at 1.0. The wide differences in symptomatology of diabetic and hypoglycemic coma lend support to a difference in etiology of the two.

It is indeed doubtful that diabetic coma can develop in the absence of ketosis. The ketone bodies certainly cannot be excused from an important etiologic role in diabetic coma simply because they may be present at times without the occurrence of full blown diabetic coma. Their role in producing the disturbed acid-base relationship, acidosis and dehydration is well known. At least acetoacetic acid is believed to be tonic. The depressed consciousness which may occur in the clinical syndrome we call diabetic coma is probably the result of several interdependent metabolic derangements, the sum of which, rather than anyone singly, is responsible.

Ketosis apparently is not due to an inability of the tissues to oxidize glucose but rather seems to occur in conditions in which liver glycogen is seriously depleted. Thus it is a finding in starvation and in phlorhizin diabetes, in which liver glycogen drops very low and energy requirements must be supplied largely by utilization of fats. Furthermore, in the depancreatized animal large doses of glucose administered

intravenously will decrease ketonemia and and ketonureas, concomitant with storage of glycogen in the liver in spite of the fact that glucose probably is not being burned by the peripheral tissues.

Ketosis is not a finding in hypoglycemia, except in rare instances, as liver glycogen is not depleted in this condition. It is a rare possibility in renal diabetes with a very low renal glucose threshold and in von Gierke's disease. In the former condition, depletion of liver glycogen occurs before ketonuria and hypoglycemia appear, and in the latter the liver, though filled with glycogen, is functionally without glycogen, as the glycogen present cannot be utilized. Infiltration of the liver with fat in the latter condition is common in the glycogen depleted liver.

The question whether insulin acts to produce an active, gamma glucose is still completely hypothetical. As far as can be determined, there is no definite evidence on this point.—*J.A.M.A.*

**Psoas muscle syndrome.**—The more common subjective manifestation of psoas myositis and fibrositis syndrome is unilateral or bilateral dull aching pain in the lower quadrant of the abdomen, which may persist for days, weeks, months, and even years. Exacerbations and remissions with complete disappearance of symptoms are common. Tenderness over the psoas muscle is the only definite objective manifestation. This tenderness is increased when the muscle is contracted or stretched, and as a rule extends from the origin of the muscle along its entire course to the tendon and to its insertion into the femur. Both psoas muscles are frequently involved, but one side is usually the more severe. Trauma, defects in body posture, acute infections, or pregnancy appear to be exciting causes. Sacroiliac syndrome and fibrositis of other muscles are common accompaniments. The syndrome may be confused with diseases of the appendix, small bowel, colon, urinary tract, uterus, fallopian tubes, ovaries and vertebral column. In 25 of the 50 cases abdominal operations have been performed alone or in conjunction with perineal repair because of mistaken diagnosis.—*GREENE, J. A.: Syndrome of psoas myositis and fibrositis: its manifestations and its significance in differential diagnosis of lower abdominal pain. Ann. Int. Med. 23: 30-34, July 1945.—Na Med. Bull.*

**Rubbing the skin after subcutaneous injections.**—The object of this manoeuvre is to disperse the injected fluid and so hasten its absorption. This may be of slight benefit when the fluid is a drug which is required to act as quickly as possible. It is actually contra-indicated after the injection of an active immunizing agent, particularly A. P. J. In other circumstances, it is merely superfluous. The added risk of infection must be very slight; pressure would tend to produce an outward flow along the track of the needle, and penetration by bacteria

from the surface should therefore not occur. Adequate treatment with iodine or spirit does disinfect the skins in the sense of ridding it to a large extent of its 'transient' and possibly dangerous flora. It is impossible to destroy the 'resident' flora; (including staphylococci), but then organisms are usually non-pathogenic.—*B.M.J.*

**Supra-orbital neuralgia.**—Supra-orbital neuralgia is a term used to describe more than one condition. It may mean a pain, strictly confined to the distribution of the supra-orbital nerve or it may include any pain situated above the orbit. Supra-orbital neuralgia in the former sense is likely to be associated with some tenderness of the supra-orbital nerve, and often with hyperaesthesia and hyperalgesia of the skins which it supplies. It is due to toxic or inflammatory lesions of the nerve itself, and is likely to be relieved temporarily by an injection of novocaine into the nerve, and permanently by following this with a few minims of 90% alcohol.

Supra-orbital neuralgia in the more general sense may be a symptom of a large variety of disorders. The pain may be referred to this region from the eye, nasal sinuses, and the upper incisor teeth. One form of supra-orbital neuralgia is associated with migraine and is known as migrainous neuralgia. Trigeminal neuralgia usually involves the ophthalmic division only after the second or third division is already affected. Lastly, it must be remembered that such pain is sometimes psychogenic. The treatment will depend on the cause.—*B.M.J.*

**Contra-indications to the use of dicumarol.**—Dicumarol is contra-indicated in: (1) the presence of definite renal insufficiency, because renal insufficiency greatly prolongs and increases its effect; (2) the presence of definite hepatic insufficiency for the same reason; (3) purpura of any type

because of the danger of bleeding when capillary weakness and impaired coagulation are both present; (4) subacute bacterial endocarditis because of the vascular weakness caused by the disease and therefore increased liability to haemorrhage; (5) blood dyscrasia with tendency to bleed; and (6) recent operation on the brain or spinal cord because of the grave consequence of even slight bleeding at the operative site.—*BARKER, N. W.: Clinical use of dicumarol. M. Clin. North America 29: 929-935, July 1945.—Na. Med. Bull.*

**Penicillin in varicose ulcers.**—Highly satisfactory results from the use of local applications of penicillin in "chronic stasis ulcers" of the lower extremities are reported by D.L. Lovell (*Archives of Surgery*, July-August, 1945, 51, 22). In most of the cases the predominant organism was *Staph. aureus*. The ulcers were cleaned, and a gauze dressing saturated with isotonic solution of sodium chloride containing 500 units of the sodium salt of penicillin per c.cm. was applied directly to the ulcer. A Unna paste boot was then applied extending from the base of the toes to just below the knee. Whenever possible the dressing was changed twice weekly, although in some instances this was only possible once a week. In four cases previous treatment with sulphathiazole had failed to effect a cure. Rapid healing (ten days to ten weeks) occurred in the five cases of early ulceration. One patient did not complete the course of treatment, but when last seen the ulcer was healing satisfactorily. In the remaining four cases the infection was soon controlled, but healing was slow, presumably on account of the defective local blood supply. It is considered that such chronic cases should be treated by radical excision of the ulcer and covering of the defect with a skin graft, unless operation is contra-indicated. Penicillin would thus appear to be superior to other bacteriostatic agents generally used for this purpose.—*Practitioner*.

## SURGERY

**Gas gangrene.**—Gledhill reviews observations on 33 cases of gas gangrene that were seen at a forward general hospital in Italy between September and November 1944. During the period under review 2951 battle casualties were admitted to the hospital. Thus the incidence of gas gangrene was 11 per thousand. Three clinical types of gas gangrene were seen. Gas abscess, which was observed in 6 cases, was characterized by a swollen tense painful limb with localized edema and induration. On incision a gush of foul smelling pus and gas escaped from a cavity in which a foreign body was often lodged. Response to drainage chemotherapy was rapid. In clostridial myositis, which was observed in 17 cases, extensive necrosis of muscle was always present with swelling, induration and frequently crepitation. Isolated muscle groups were rarely involved alone. More often there was a

widespread infection of all the muscles along the track of the missile. There was severe general toxemia. The 10 cases of fulminating gas gangrene presented a picture of grave toxemia. The local signs were those of severe clostridial myositis with the viability of the limb often in doubt. The general condition varied from semi-coma to muttering delirium. In the treatment of gas gangrene, early surgical removal of the source of toxemia is still the basis of treatment. Penicillin used prophylactically, both locally and parenterally, did not prevent the onset of gas gangrene. In treatment penicillin appears to be of extreme value. The usual dosage locally was a total of 17,600 Oxford units per tube. From 300,000 to 500,000 units was average parenteral dose per case. Most patients were given a course of from 25 to 30 Gm. of sulfathiazole. A few had intravenous sulfadiazine or local sulfonamide

powder. Every patient was given from 2 to 4 pints of whole blood and gas gangrene anti-serum. The efficacy of the combined use of surgery, penicillin, sulfonamides, blood transfusion and serum was proved by the fact that only one death occurred among the 33 patients, and this occurred after the clostridial infection has been controlled.—J. A. M. A.

**Recurrent paroxysmal abdominal pain.**—Transient acute abdominal pain affecting an apparently healthy person is often difficult to explain; if the symptoms are of short duration, and if operation is clearly contraindicated, precise diagnosis may be impossible. From time to time, however, little groups of cases are separated from the mysterious majority, and the number of the unexplained grows gradually less. Attacks of abdominal pain have long been known in subjects of erythema and of purpura. Siegal has now attempted to distinguish in a separate clinical category an acute affection similar but free from mixed cutaneous eruption. According to Siegal this syndrome is characterized by recurrent paroxysms of severe abdominal pain, diffuse and cramp like or localized and gnawing. The attacks are said to begin as a rule in the second or third decade, and they continue for many years without affecting the patient's general health. Early attacks may be noted only once or twice a year, but later a more frequent cycle is established, the pain recurring weekly or monthly. The temperature rises during an attack, sometimes to 104° or 105°F. Nausea, vomiting, and diarrhoea may occur; the abdomen is tender, the abdominal muscles may be spasmodically contracted and rebound tenderness is sometimes elicited. There may be pain in the chest, too, and sometimes shoulder pain; prostration may be extreme, and the white count is high.

One of Siegal's patients was submitted to laboratory during an acute attack of pain, and the subserous vessels of the ileum, caecum, and sigmoid colon were seen to be congested. From this evidence and from the abdominal tenderness and rebound tenderness which are present during acute attacks, Siegal suggests that the name 'benign paroxysmal peritonitis' be applied to the condition until the aetiology and pathogenesis are better understood. There is still room for argument whether the existence of this syndrome as a distinct entity is justified, and whether, if it is justified, Siegal's title for it is suitable. But Siegal goes further, and suggests, though only tentatively, that the abdominal symptoms

are due to allergy. A family history of allergy was elicited in the majority of cases, and urticarial wheals were occasionally observed during acute attacks, but positive allergic reactions were seldom elicited, in individuals. In no case was any allergen proved capable of initiating an attack, and the hypodermic administration of adrenaline gave no relief from pain. That acute abdominal symptoms may accompany urticaria and angioneurotic oedema is certain, and there is no reason to doubt that some forms at least of paroxysmal abdominal pain may be allergic in origin, even if unaccompanied by cutaneous manifestation. It would be wrong, however, in any individual case to ascribe acute abdominal symptoms to hypersensitization unless clear evidence of allergy were obtained. The only certain proof would be the precipitation of an attack of pain by the administration of the responsible allergen. If Henock's purpura be excepted no precise account yet exists of an abdominal syndrome which, from the abdominal evidence alone, can be regarded as characteristic of, and peculiar, to, allergic state; it is equally doubtful whether the abdominal symptoms of frankly expressed allergy are due to peritonitis.—B.M.J.

**Carcinoma and digital and rectal examination.**—Digital examination exceeds and precedes in importance all other methods of examination for carcinoma of the lower sigmoid and rectum. Proctoscopy, sigmoidoscopy and X-ray examination are at times necessary but are refinements in technique. In contrast, digital examination is simple and available at all times to the physician. There is no excuse for omitting it when the patient complains of rectal bleeding. It is of more value in the diagnosis of malignancy of the rectum than any other one procedure.

The condition may often be detected by digital examination and missed by other methods in unskilled hands. With the patient in the left lateral Sim's position, hips well out on the edge of the examining table, the average index finger can easily palpate the majority of rectal lesions. There is rarely any contra-indication to this type of examination.

Digital rectal examination is the most important, the most informative and the most indispensable of all the methods employed by proctologists to determine the presence of carcinoma of the rectum. Physicians will save themselves much grief by utilizing it regularly.—J. Gerendasy, M.D., Elizabeth, N. J.—J.M.S.N.J.

## OPHTHALMOLOGY

**Myasthenia gravis and its ocular signs: A Review.**—Frank B. Walsh, *Am. J. Ophthalm.* 28: 13 (Jan.), 1945.

This study has nothing to add to what has already been written regarding the ocular signs of myasthenia gravis.

However, the following observations were made; (1) ocular symptoms and/or signs were present in all cases; (2) purely "ocular" myasthenia gravis was observed several times, but in such cases there is always the possibility of spread of the

weaknesses; (3) the ocular symptoms and signs usually appear early in the course of the disease, but occasionally they are a late development; (4) ptosis is the most constant ocular sign; (5) edema of the lids is a rare prodromal sign; (6) retraction of the lids is a rare ocular sign which was observed in one case after a chronic ptosis; (7) the similarity of abnormal associated movements of the lids in myasthenia gravis, and as a result of misdirection of regenerated fibers in the third nerve, was noted. An explanation for these lid phenomena in myasthenia gravis was not attempted; weakness of the orbicularis oculi may occur in the absence of ptosis or with it. Probably weakness in closure of the lids is overlooked more often than any other common ocular sign; (9) limitation of ocular movements occurs unilaterally or bilaterally and in almost all combinations; (10) when pupillary abnormalities are present, it is doubtful if the case is one of myasthenia gravis; (11) changes in accommodation were observed in only one case; (12) the visual fields and visual acuity are not altered; (13) the similarity of the ocular signs seen in myasthenia gravis and in thyroid states is noteworthy.—E.E.N.T.

**Penetration of penicillin into the eye.**—Penicillin was capable of traversing the

human cornea when introduced into the conjunctival sac by continuous irrigation with a solution. The damaged cornea apparently allows considerable penetration but the normal cornea is less permeable. A damaged epithelium (especially if edematous throughout) or a cocaineized epithelium probably behaves as would epithelium treated with a wetting solution. When penicillin was administered intramuscularly, it appeared in the aqueous humor of both primary and secondary types but in very much lower concentration than that attained in the serum. Higher intra-ocular concentrations than those found after intramuscular penicillin were produced in each eye subjected to iontophoresis, but the improvised apparatus employed produced an undesirable complication (corneal nebula). It is at present impossible to define the concentration of penicillin in either blood or tissue fluid necessary for therapeutic effect. It may be that the small amounts of penicillin reaching the aqueous humor after intramuscular penicillin or penicillin baths, will prove to be clinically adequate. The fact that high intra-ocular concentrations can be obtained by the iontophoresis method, is significant and probably worthy of further investigation.—WRIGHT, R. E. and STUART-HARRIS, C. H.: Penetration of Penicillin into Eye.—*Brit. J. Ophthalm.* 29: 428-436, August 1945.

## OBSTETRICS and GYNÆCOLOGY

**Perforating endometriosis of posterior fornix.**—According to Arenas and Blanchard, endometriosis of the posterior fornix may be caused by any of the following conditions: (1) endocervicitis, (2) reflux of menstrual blood, (3) rupture of an ovarian cyst, (4) implantation of decidual cells after obstetric trauma and (5) proliferation of aberrant endometrial tissue. The condition is observed, as a rule, in women between 30 and 40 years of age. The symptoms are more or less acute. There is a hypermenorrhea, intermenstrual or premenstrual bleeding, rectal pain and small rectal hemorrhages. Small, dark blue cysts and bleeding granulations partially covered by the vaginal folds are seen during the gynecologic examination. The appearance of the vaginal lesion is that of a scar. There is an early infiltration of the recto-vaginal septum and later stricture of the rectum. The diagnosis is arrived at by a biopsy. The therapy consists in irradiation of the ovaries or a panhysterectomy. In the case reported by the authors, endometriosis of the posterior fornix perforated the cul-de-sac of Douglas and infiltrated the recto-vaginal

septum. Treatment consisted in a bilateral adnexectomy.—J.A.M.A.

**Douches.**—Karnaky (*American Journal of Surgery*, July, 1945), points out that many physicians are still prescribing alkaline substances, sodium perborate, borax, sodium bicarbonate, potassium permanganate, magnesium sulphate and sodium chloride or a combination of these, in the treatment of vaginal leukorrhea or as a cleansing douche when it is scientifically established that the normal hydrogen ion concentration of the adult vagina is acid. He thinks that acid douches (ph 3.0 to 5.0) should be used, because with pathologic changes there is hypoacidity of the vagina, and, as these changes increase, the hydrogen ion concentration approaches the alkaline side. Alkaline douches favor the growth of pathogenic organisms. The best douche found in ten years of research is vinegar. The patient should be instructed to use 5 tablespoons of vinegar to the douche bag if there is an acute vaginitis. After two weeks, 2 to 3 tablespoons is used instead of 5. For an ordinary cleansing douche 3 tablespoons of vinegar should be added to the douche bag.—G.P.

## A Query

**Can head-injury cause deafness after an expiry of 2½ years?**

I have got a case of a young man of 24 who received a head injury in June 1942 with some affection of the left 3rd cranial nerve. He was discharged cured from the

hospital after a couple of months. He was physically fit in all respects till in January 1945 he got bilateral acute otitis etc., and also complained of deafness. He was treated with sulphonamide dusting after mopping up the discharge with a swab. The

discharge cleared off in about a week but the deafness did not improve. He was referred to an E. N. T. specialist who opined that the deafness was not due to any aural condition. He was then referred to a psychiatrist for opinion regarding the presence of hysterical element.

His report was also negative.

Can anybody suggest any diagnosis or treatment?

138, Ind. Rly. Const.  
Coy. I. E., Calcutta,  
12, Army P.O.,

A. L. WAHI,  
Capt., I.A.M.C.

### Correspondence

#### Princess Tsahai Memorial Hospital

AN APPEAL FROM EMINENT PHYSICIANS,  
SURGEONS AND HOSPITAL MATRONS

To The Editors, THE ANTISEPTIC, Madras.  
Sirs,

During the years 1936-1941, in which Italy exerted uneasy rule over Ethiopia, whose liberties have now been regained by the aid of Britain, the Emperor of Ethiopia lived in this country. His daughter, Princess Tsahai, undertook training as a nurse at the Great Ormond Street Hospital, for Sick Children and, after passing the examination qualifying her to become a State Registered Nurse, she entered Guy's Hospital for further training. Her object was to help the country which had given her hospitality and also to fit herself to lead the movement for a modern medical and nursing service in Ethiopia, after its freedom had been restored for she never doubted that right would triumph in the end. When this time came she returned to her country with the nucleus of the organisation she intended to create; but most tragically, her great work was stopped by her illness and death at the early age of 22, on August 11th, 1942, only a year after her return home.

An appeal is being made for funds to found in Ethiopia, a Hospital with Medical School and Nursing School, Library and Ambulance Services, in memory of the Princess Tsahai, and as a token of good-will to Ethiopia from the people of Great Britain.

H. I. M. the Emperor of Ethiopia made available to the Memorial Council a partially constructed hospital, situated on the outskirts of Addis Ababa. It is surrounded by magnificent country, with ample grounds and space for extension. The first unit will comprise Administrative Offices, General Hospital Children's Section and out-patients department. The treatment and study of tropical diseases will occupy a major share in the activities of the hospital. Ethiopia offers a particularly varied field for such work.

A medical and nursing school is greatly needed, and the Memorial Hospital aims at furnishing this essential requirement. Nursing in Ethiopian Hospitals is now done by untrained dressers, poor people often lacking even elementary education. The Nursing Profession has yet to be created. There are no wards for sick children.

A target of £100,000, was set for the hospital fund, of which the first half has been raised, and for which a further effort must now be made.

### A Correction

In line 17, Page 568 of October '45 issue of the ANTISEPTIC, please read 0.5 c.c. for o.s.c.c.

Those who are engaged in Health and Hospital Services of Great Britain are responding generously to this appeal. We are confident of not less generous assistance from the Medical and Nursing Professions of the Empire. The gifts of the Nursing and Medical Professions will be commemorated in some special part of the hospital.

Please draw and cross cheques and postal orders in favour of "Princess Tsahai Memorial Hospital Fund," and address them to Lord Horder, Hon. Treasurer, Princess Tsahai Memorial Hospital Fund, c/o H. Reynolds & Co., Chartered Accountants, 1, Bloomsbury Court, High Holborn, W.C.1.

The undersigned cordially endorse this appeal to Medical and Nursing Professions of Great Britain. Yours etc.

E. Farquhar Buzzard, Bt. K.C.V.O., M.A., M.D., F.R.C.P., Christ Church, University of Oxford.

H. W. Hetherington, M.A., LL.D., Vice-Chancellor, University of Glasgow.

Arnold D. McNair, C.B.E., F.B.A., LL.D. Vice-Chancellor, University of Liverpool.

Charles McNeil, M.A., M.D., Ch. B., F.R.C.P., Ed., Past President of the Royal College of Physicians of Edinburgh.

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J. W. Struthers, M.B., Ch. B., F.R.C.S., Ed. President of the Royal College of Surgeons, Ed.

Moran, M.C., M.D., President of the Royal College of Physicians of London.

Alfred Webb-Johnson, Bt., K.C.V.O., F.R.C.S., F.A.C.S., President of the Royal College of Surgeons of England.

#### POSTSCRIPT FROM HOSPITAL MATRONS.

This Fund makes a warm appeal to the Nurses of Britain. It should be remembered that the Princess Tsahai was a nurse. She realised to the full the great importance of the Nurse's mission in the healing of her people, and the essential part of the Nurse in all Hospital Services. She had already proved both the value of her British Hospital training and her own courage and initiative by the work she inaugurated on her return home.

The Memorial will stimulate her compatriots to similar service. We are glad to add our word of support:

Dorothy, A. Lane, Matron, The Hospital for Sick children.

Emily E. Macmanus, Matron, Guy's Hospital.

Lina Williams, Matron, Royal National Orthopaedic Hospital.

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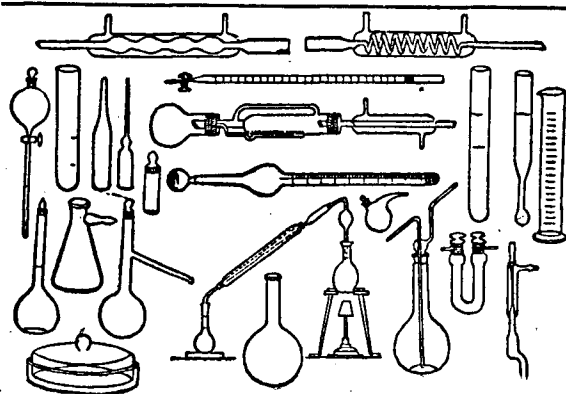
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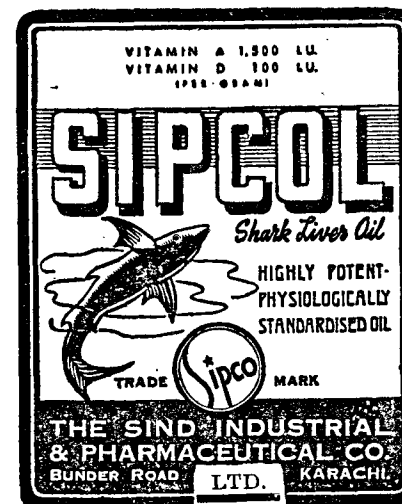
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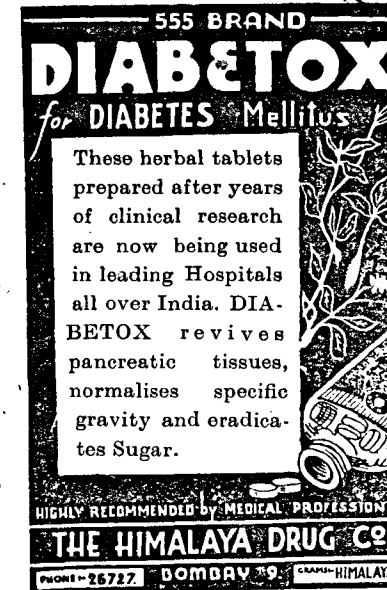
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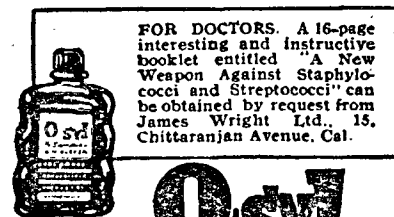
Each finger was then used as a "test", for which purpose it was first immersed in the fluid under examination for the required length of time, transferred to a bowl of distilled water for a period of one half-minute to remove traces of any disinfectant, etc., after which a dry sterile wool swab was rubbed vigorously over the finger tip for a period of one half-minute (timed).

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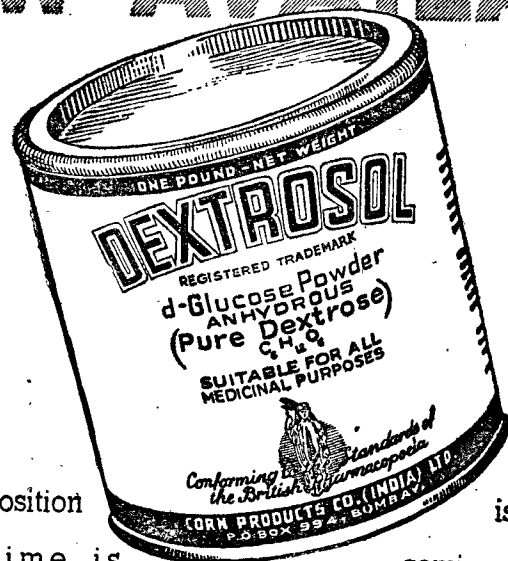
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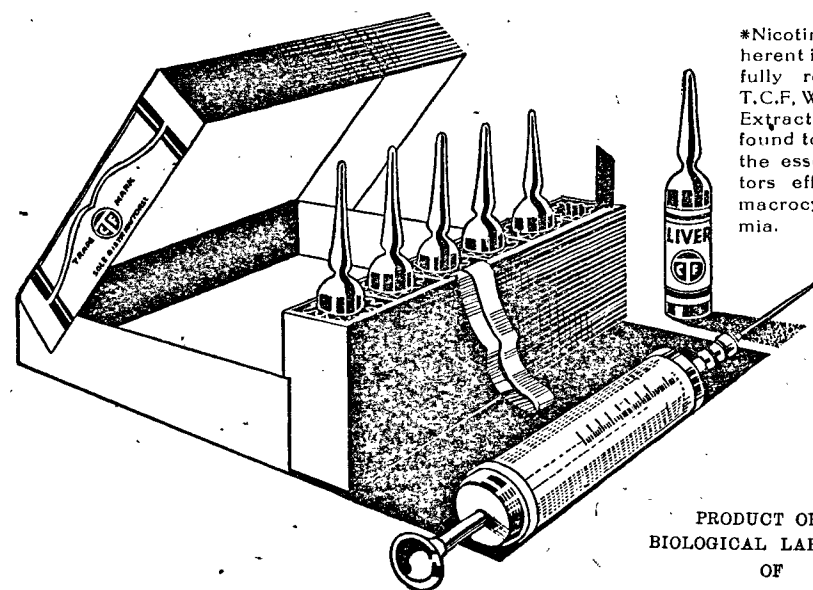
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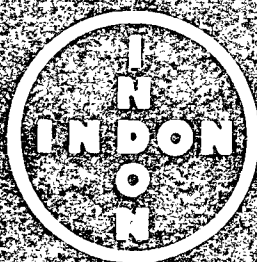
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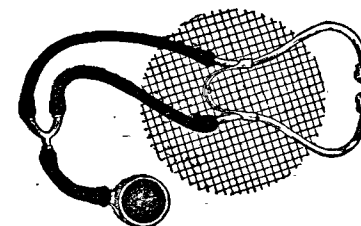
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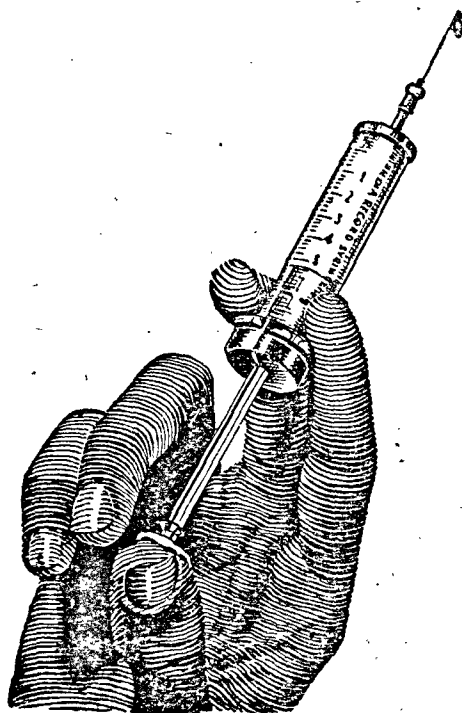
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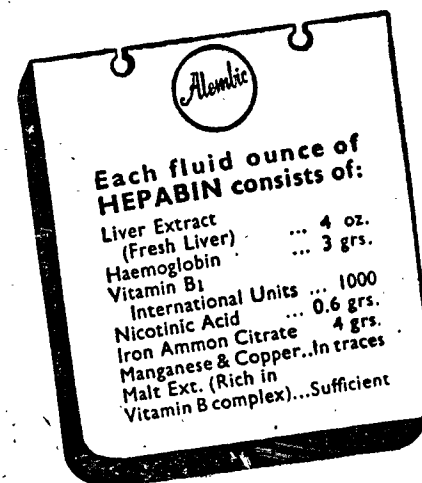
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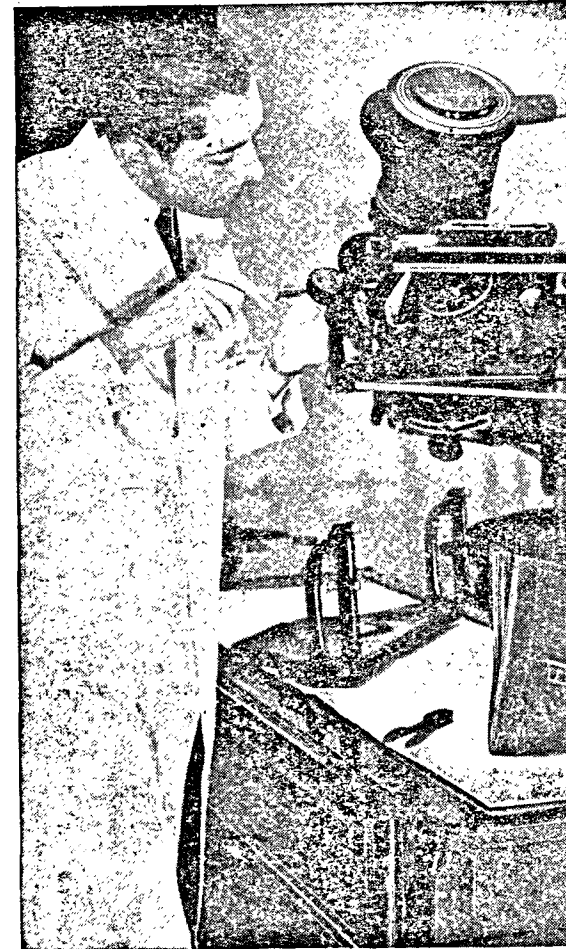
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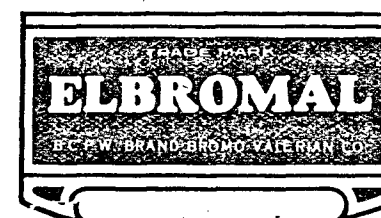
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