The presence of cancer cells growing in the epidermis leads to a definite reaction in the cutis. There occurs new formation of capillaries, and this is accompanied by extensive infiltration of leucocytes and plasma cells; I have also met with eosinophils in large numbers. When the growth within the epidermis is discontinuous, a definite relationship between the affection of the epidermis and the underlying cellular reaction can often be recognized. Owing to the new formation of blood vessels and their congested state, and to a certain amount of exudate which soaks through the epidermis, the characteristic features of such lesions are produced. Of course, at a later stage the affected epidermis may become destroyed, and then there is a red surface formed by granulation tissue.

BIBLIOGRAPHY.

- Borrmann, R.: *Zeit. f. Krebsforsch.*, 1904, ii, 1.
 Borst: *Verhand. d. deutsch. path. Gesellsch.*, 1904, Tag. vii, 118.
 Bowen, J. F.: *Journ. Cutan. Dis.*, 1912, xxx, 241.
 Charteris, A. A.: *Journ. Path. and Bact.*, 1930, xxxiii, 101.
 Cheattle, G. L.: *Brit. Journ. of Surg.*, 1926, xiii, 509.
 Dunn, J. Shaw: *Journ. Path. and Bact.*, 1930, xxxiii, 297.
 Eller, J. J., and Anderson, M. P.: *Brit. Journ. of Dermatol. and Syph.*, 1930, xlii, 263.
 Fraser, J. F.: *Arch. Derm. and Syph.*, 1928, xviii, 809.
 Jacobaeus, H. C.: *Virchow's Archiv*, 1904, clxxviii, 124.
 Muir, R.: *Journ. Path. and Bact.*, 1927, xxx, 451.
 Murray, J. A., and Woglom, W. H.: *Scientific Reports of the Imperial Cancer Research Fund*, 1921, vii, 45.
 Simard, Ch.: *Bull. Assoc. Franç. pour l'Étude du Cancer*, 1930, xix, 50.
 Susman, M. P.: *Brit. Journ. of Surg.*, 1927-28, xv, 635.
 Whitfield, A.: *Brit. Journ. of Dermatol. and Syph.*, 1929, xli, 177.
 Wise, F.: *Arch. Derm. and Syph.*, 1929, xix, 1.

EFFECTS OF HYPERTONIC SALINE IN THE TOXAEMIAS OF LATER PREGNANCY.*

BY

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Four years ago the authors, in a paper entitled "Diet in the treatment of pre-eclampsia," advocated the use of salt-free diet.¹ Such diets contained about 3 grams of chloride reckoned as sodium chloride, and are more usually nowadays, and more legitimately, called salt-poor. At the time the authors were particularly concerned in making clear that diets high in protein or in fat were quite innocuous to pre-eclampsia. As a practical corollary it followed that ordinary mixed diets of protein, carbohydrate, and fat could be utilized, provided they were salt-free or salt-poor. Not only were such diets innocuous to the pathological condition, but the patients showed marked clinical improvement. In view of the number of factors concerned, however, we did not consider it legitimate to ascribe the improvement to the protein or fat in the diet. Rest in bed and general attention to hygiene are such powerful factors in general clinical improvement that it required much stronger evidence than we possessed to conclude that high protein feeding or high fat feeding was beneficial.

We were also interested in the effect of our observations upon theories of etiology. Protein *per se*—if our observations were correct—could not play a part in the production of eclampsia. Those authors who had ascribed the reduction in the incidence of eclampsia in Germany² during the war as due to a reduction of dietary protein could not, in our opinion, have been correct. That a dietary factor exists appears certain. That the dietary factor is protein, or even fat, is uncertain, despite the popularity of the

former supposition. We may state in further support of our contention that since our previous publication we have given high protein diets, not to pre-eclampsia, in whom there might be a doubt that actual convulsions would occur, but to eclampsia, who, having responded to treatment with sedatives, were admitted to the ward for further observations. We noticed no aggravation of symptoms; indeed, as previously, we found an improvement.

The use of salt-free or salt-poor diets in the treatment of nephritis of the parenchymatous type is well known. The influence of sodium chloride or sodium bicarbonate in definitely aggravating the symptoms of albuminuria and oedema in that condition is a matter of well-known clinical and laboratory demonstration. The observations of our previous paper may have been open to the criticism that we were dealing with patients in whom the kidney lesions were of the parenchymatous type. De Wesselow³ has emphasized the occurrence of this type in the toxæmias of pregnancy.

Since then, however, we have been able to reinforce our conclusions on the part played by sodium chloride by a series of further observations—namely, by the use of sodium chloride intravenously. The use of hypertonic saline solutions has become very general in some centres for the treatment of post-operative paralytic ileus, either actual or threatened. It is also a powerful dehydrating agent and diuretic. In the treatment of eclampsia or threatened eclampsia one notices, too, a tendency towards the use of intravenously administered dehydrating agents. Lazard⁴ has enthusiastically advocated the use of intravenous magnesium sulphate. Titus⁵ uses 50 per cent. glucose solution, though with a confessedly different object. Mussey⁶ recommends ammonium chloride by the mouth. A brief review of many of the older methods of treatment shows that some of the therapeutic agents removed water—for example, purging, venesection, hot packs. These were used frequently and often with success. Their object was to remove toxin, but at the same time they removed body fluid. Thus the use of hypertonic saline solutions might prove beneficial. True it might produce or aggravate oedema in a case complicated by parenchymatous nephritis, but the symptom of subcutaneous oedema has never been considered as the dangerous one in an eclamptic or pre-eclamptic patient. To relieve the symptoms of convulsions and high blood pressure, with its attendant dangers of haemorrhage, would be well worth the cost of a little oedema.

These latter arguments, valid as they may sound, are fallacious. The use of hypertonic saline solutions in toxæmias of later pregnancy is harmful. We present four cases, one normal acting as a control, the other three being varying degrees of toxæmias, though we feel some doubt as to the diagnosis in Case II.

CASE I.—Normal Pregnancy. 0 Para. 8 Months Pregnant.
Aged 19.

Date.	Flood Pressure (mm.).	Urine Albumin (mg. per 100 c.cm.).	Notes.
1929 June 6	128/64	0	Full diet.
" 7			"
" 8	120/58	0	"
" 9			
" 10	124/68	0	300 c.cm. of 10% saline intravenous.
" 11	124/53	0	" "
" 12	117/50	0	Full diet.

The patient had been observed carefully in the clinic, and was normal. The intravenous saline was given on June 10th at 2 p.m. after the noon meal. She complained of thirst, and was given chipped ice; she took an ordinary supper. On June 11th the intravenous saline was given at 11 a.m., and the patient fed on orange juice the remainder of the day. Later in the day two fluid bowel movements occurred, but otherwise no effects were observed.

* A paper read in opening a discussion in the Section of Obstetrics at the Annual Meeting of the British Medical Association, Winnipeg, 1930.

CASE II.—Possibly Mild Toxaemia. 4-Para. 8½ Months Pregnant. Aged 22.

Date.	Blood Pressure (mm.).	Urine Albumin (mg. per 100 c.cm.).	Notes.
1929 March 15	156/92	50	Few pus cells in urine. Full diet. Pot. cit. 30 grains. Sod. bicarb. 30 grains. Two-hourly.
" 16	138/98	20	
" 17	128/90	10	
" 18	134/90	30	370 c.cm. 10% saline intravenous.
" 19	117/90	10	Complains of nausea. Milk diet.
" 20	118/84	10	Headache. Milk diet.
" 21	118/84	20	Well. Milk diet.

The patient was admitted from the outdoor clinic as suffering from mild toxaemia. She had slight puffiness in the ankles and face, and a blood pressure of 150 mm. A trace of albumin was found. She improved at once with rest in bed. The intravenous saline, given at 2 p.m., caused excessive thirst, and the patient complained of weakness. Chipped ice was given to allay the thirst. Nausea and headache were complained of on the two following days. There was no oedema. A successful medical induction was carried out the following week.

CASE III.—Mild Toxaemia. 1-Para. 8½ Months Pregnant. Aged 21.

Date.	Blood Pressure (mm.).	Urine Albumin (mg. per 100 c.cm.).	Notes.
1929 Feb. 26	140/95	200	Full diet.
" 27	120/96	190	300 c.cm. 10% saline intravenous 2 p.m. Milk diet. Vomiting.
" 28	134/30	250	Backache.
March 1	150/110	450	Backache. Vomiting.
" 2	138/80	—	Labour.

The patient was admitted from the pre-natal clinic on account of irregular gain in weight, slight increases in blood pressure, and sudden albuminuria. A stillborn birth had followed a previous pregnancy. The patient felt perfectly well; there were no headaches nor oedema. When the intravenous saline had been given the increase in albuminuria was marked. The blood pressure, both systolic and diastolic, on March 1st was the highest recorded over a long series of observations. Labour was prolonged, lasting three days. The baby was normal. On March 6th the blood pressure was 130/90, and the albuminuria was 100 mg. per 100 c.cm.

CASE IV.—0-Para. 8 Months Pregnant. Aged 17.

Date.	Blood Pressure (mm.).	Urine Albumin (mg. per 100 c.cm.).	Notes.
1929. Nov. 16	190/130	—	Admitted one convulsion. No oedema. Stroganoff treatment. Improved. Milk diet.
" 17		900	
" 18	140/112		Salt-free mixed diet.
" 19	130/110	650	Feeling well.
" 20	150/110		
" 21	148/114		
" 22	134/104	500	
" 23	122/90	400	
" 24	120/98	350	
" 25	115/84	200	
" 26 a.m.	128/100	250	300 c.cm. 6% saline intravenous 9 a.m.
" p.m.	135/115	375	
" 27 a.m.	130/115		370 c.cm. 10% saline intravenous 10 a.m.
" noon	178/158		Headache. Epigastric pain. Vomited.
" 3 p.m.	160/120		Vomited. Face congested. No oedema.
" 6 p.m.	180/118		Seems a little improved.
" 9 p.m.	200/140		Convulsions. Stroganoff treatment. Strong uterine contractions. Labour 11 p.m.
" 28	130/98	800	Water only.
" 29	140/100		Milk diet.
" 30	130/98		"
Dec. 1	130/100	200	Feeling well.

The effect of the intravenous saline solution on the blood pressure, albuminuria, and general symptoms was very clear. A colon irrigation preceding the labour was followed by a veritable deluge of fluid. The labour was very precipitate. The foetus was small and ill developed.

Two well-defined criteria for estimating the extent of a toxaemia of later pregnancy are blood pressure and albuminuria. Judged by these standards our observations divide themselves into two groups. The first, consisting of Cases I and II—where Case I is normal and Case II is a doubtful toxaemia—show no effect at all. Both patients had thirst, as would be expected in any normal individual. In Case I there were two fluid bowel movements, and the patient in Case II complained of nausea and headache on the two days following the injection. The elimination of salt and water by the bowel was in all probability the reason for the two fluid stools in Case I. The nausea and slight retching in Case II may be a variation of the same method of elimination which failed to relieve itself by the emptying of the bowels. The headache followed the intravenous saline and lasted two days. Judged by the major criteria of albuminuria and blood pressure, however, both cases showed no effect—indeed, Case II showed an obvious drop in pressure, despite the disturbance caused by the nausea and the headache.

Judged by the same two criteria Cases III and IV form another group. In both the albuminuria increased. In Case III the concentration of 450 mg. per 100 c.cm. urine was the highest noted throughout the whole period of observation. One of us had made fortnightly weighings and blood pressure and urinary examinations of this patient for a previous three months. Albuminuria and a slight elevation of blood pressure had been observed a fortnight previous, and on their continuation the patient was brought into hospital. Just as a peak in the albuminuria was noticed after the intravenous saline has been given, so a peak in the blood pressure level was observed, both systolic and diastolic. It is to be noticed that vomiting was present in this case also, and persisted for two days. It is hard to escape the conclusion that the hypertonic saline had aggravated the toxaemia. In Case IV the conclusion is irresistible. The changes follow one another with such dramatic suddenness that we have no difficulty in distinguishing cause and effect. On admission she had high blood pressure, marked albuminuria, and one convulsion. These, together with the absence of any history of nephritis, her age, and primiparity, make a clear diagnosis of eclampsia. The convulsive stage was easily checked by sedatives. Rest in bed and a salt-free mixed diet rapidly reduced the albuminuria and blood pressure. The effect of 48 grams of sodium chloride given in two doses on the mornings of November 25th and 27th immediately changed the whole picture. The changes were possibly evident on the evening of the first day when the blood pressure and albuminuria increased over those of the three previous days. The changes at the end of the second day represented a return to the symptoms of eclampsia. The blood pressure reached 200/140. Headache, epigastric pain, and vomiting were present. There was a typical appearance in the facies of congestion and coarseness, but no definite puffiness or pitting on pressure was observable in any part of the body. By 9.30 p.m. three convulsions had occurred. Sedatives were administered at once, but the patient went into precipitate labour, and delivered a living child within an hour. The albuminuria also had more than doubled in intensity.

From the standpoint of practical therapy the results are wellnigh disastrous. We have, at present, no wish to continue our observation in this direction. In our very incomplete state of knowledge it appears fraught with too much danger to the patient, though the very acuteness of the disturbance brought about by an external agency possibly offers safeguards of its own, of which we are only dimly aware. We refer to the onset of labour. In both of the latter cases labour supervened shortly after the disturbance initiated by the saline solution. In both cases a living child was obtained. With birth, as is often the case in rapidly developing eclampsia, there was a rapid subsidence of symptoms and a return to a normal condition. There is, however, a definite negative value to our observations—namely, the contraindication of hypertonic saline solutions in post-operative work in pregnancy where there is any indication of toxaemia, unless the pregnancy is to be terminated simultaneously.

The observations recorded in this paper extend and reinforce those of our previous work on this subject. The

commonly occurring dietetic factor which can enter into the production of eclampsia or pre-eclampsia is sodium chloride. Other observers have emphasized the value of restriction of salt in the treatment of such toxæmias from the standpoint of kidney insufficiency. Eckelt⁷ in Germany, de Wesselow and Wyatt⁸ in England, Bland and Bernstein⁹ in America, have advocated salt restriction from this standpoint. Stander¹⁰ in his recent monograph recommends a salt-free diet if there is marked oedema. In our previous paper we were inclined to think that salt could play a part in the more fundamental changes connected with the toxæmia, and suggested a possible value in salt restriction from the standpoint of pre-natal care. The observations recorded in this paper lead us to the conclusion that a high intake of salt, taken at the right moment in a developing toxæmia, will produce an albuminuria, an increased blood pressure, and convulsions, in a short period of time. Such a picture represents a fulminating eclampsia. One hears often of cases in which a patient, under good pre-natal care and with a normal blood pressure and urine, develops convulsions within a week or a fortnight; or where a patient, with a mild toxæmia, and under observation, develops convulsions within twenty-four or forty-eight hours of an office visit, when no immediate danger was suspected. If such a patient has had in the meantime a generous meal in which meat is prominent, the protein is promptly blamed. We would suggest a strict inquiry into the amount of salt taken with the protein and the usual accessories of a generous meal. Salt restriction, in our minds, is a necessary part of pre-natal care. Protein is harmless. Bland and Bernstein claimed a diminution in blood pressure when their toxæmia patients were placed on salt-free diet. This followed a three-day period of ordinary hospital diet in which no diminution in blood pressure was observed. Three days after admission to hospital is a short time in which to observe a fall in blood pressure in a case of toxæmia, even if it is accompanied by rest in bed. A week or ten days is not too short in many cases. Bland and Bernstein appear to realize the weakness of their position, for they endeavour to make the observations more equable by allowing the patients the liberty of the ward during the salt-free diet period. It is unfortunate that they did not reverse the order of their diets in half their observations.

In our previous paper we noted observations on the effect of sodium bicarbonate in a toxæmia of pregnancy. There was a recurrence of the oedema which had subsided on the salt-free diet, and a distinct rise in blood pressure both systolic and diastolic. At the time we did not wish to associate the rise in blood pressure with the sodium bicarbonate. We were more inclined to ascribe it to the nervousness of the patient herself, who had observed and knew the significance of the return of the oedema. In the light of our new observations the rise in blood pressure, following sodium bicarbonate administration in toxæmia of pregnancy, may be as justly ascribed to the retention of the sodium salt as it is after the intravenously administered sodium chloride, though it is to the sodium ion, rather than the chloride or bicarbonate ion, that we attach importance.

Our observations, too, have a bearing on the general question of a relationship between chloride retention and hypertension as claimed by Ambard and his school. On this continent Allen¹¹ championed the beneficial effect of extreme chloride retention on essential hypertension. His first observations were not substantiated by all later workers,¹² and Allen¹³ himself later realized that all classes of hypertension were not benefited by the restriction. The high blood pressure of the toxæmias of pregnancy, where functional rather than morphological changes are involved, would seem a much more promising condition in which to show the effects of a salt restriction.

From the standpoint of etiology we believe our results to be of value. It is impossible in the present state of our knowledge to separate the influence of sodium salts and water from either a physiological or a pathological point of view. Our observations in this and in the previous paper have shown a connexion between sodium chloride and convulsions, albuminuria, high blood pressure, oedema, epigastric pain, and vomiting in the toxæmic subject. All these are, or can be, symptomatic of eclampsia or pre-eclampsia. We cannot but fail to conclude that underlying

a variety of symptoms lies a unity of disturbance—a water retention, or a change in its distribution in the body. Zangemeister¹⁴ has stressed the importance of observing the gains in weight during pregnancy and of suspecting any abnormally high gain as a sign of impending toxæmia. Ginglinger¹⁵ judges his toxæmia cases by the Cl retention, the daily weight of the patient, and a measurement of the water intake and output. Holtermann¹⁶ tests for latent oedema in pregnancy by Kaufmann's modification of Frey's diuresis test. Siderow,¹⁷ using Galeotti's method of measuring insensible perspiration, finds a very low output in toxæmias.

Our conception of the toxæmias of later pregnancy is a disturbance involving fundamentally a change in water distribution. This may be preceded or accompanied by a water retention showing itself in an abnormal gain in weight (Zangemeister). Once initiated, however, the disturbance may show itself in many ways: (a) epigastric pain and vomiting—the significance of an attack of "acute indigestion" as symptomatic of a toxæmia when occurring in later pregnancy has often been emphasized; (b) albuminuria—with a kidney lesion of the parenchymatous type or mixed; (c) hypertension—often present as the sole symptom, or accompanied by a slight albuminuria, in pregnancy of middle-aged multiparae; (d) oedema—one of the commonest of symptoms; (e) convulsions—occurring by themselves these resemble epileptic seizures, and can only be distinguished by the fact of pregnancy and the absence of any previous history of epilepsy; (f) perilobular liver degeneration—not always present.¹⁸ One, or many, or any combination of these symptoms may accompany the water disturbance. Herrick¹⁹ has pointed out how the constitution or the make-up (possibly endocrine) of the mother determines the course of the toxæmia as it shows itself in successive pregnancies. In our opinion it is this maternal constitution which determines the appearance of one or any combination of symptoms.

We have referred to changes in water distribution as well as to water retention. An interesting case, illustrating a relationship between oedema and high blood pressure, has been referred to us by our colleague Dr. W. G. Cosbie, who has kindly allowed us to make use of his notes. The patient had been under his care and routine observation from the beginning of her pregnancy and had been perfectly well until February 19th, when she complained of a severe headache. The essential symptoms and observations are best given in chart form.

CASE V.—O-Para. 5 Months Pregnant. Aged 31.

Date.	Blood Pressure (mm.).	Urine Albumin.	Oedema.	Notes.
1929. Feb. 14	136	0	0	Well.
" 19	150	0	++	Severe headache. Oedema in face and feet. Bed with salt-free diet.
" 20	170	+	+	Milk diet only. Oedema less. No headache.
" 21	190	++	0	Severe occipital and frontal headache. No oedema. One hour after record of B.P. two convulsions. Modified Stroganoff treatment.
" 22	142	+++	++++	General anasarca.
" 23	160		++	Oedema disappearing. Hot pack.
" 24	140	+	+++	Oedema returning.
" 25	160	++	+	Oedema disappearing.
" 26	170	+++	+	Oedema very slight.
" 27	196	++++	0	No oedema. Uterus emptied.

The sudden onset of symptoms was marked by a moderate rise in blood pressure, with a moderate oedema. In two days, in spite of a salt-poor diet and a milk diet, the blood pressure mounted to the convulsive level, with a disappearance of the oedema. In twenty-four hours the blood pressure had fallen and given place to a massive general oedema. The treatment on February 21st included magnesium sulphate to ensure brisk elimination by the bowel. It seems reasonable to suppose that the water in the oedema had

come from the cells, since little or none had been given by the mouth, and that the water in the cells had been responsible for the high blood pressure. An opposite state of affairs would appear to have taken place between February 24th and 27th. The oedema subsided and the blood pressure rose. Apprehensive of further convulsions, the uterus was emptied on February 27th. The increase in the albuminuria during the same period is also significant.

Allusions are to be found in the literature to the similarity of the convulsions of eclampsia to those produced in animals by the continuous forced administration of water. Rowntree and his co-workers²⁰ discovered and investigated this latter phenomenon and termed it "water-intoxication." It is characterized by a positive water balance, but this is correlated with an alteration in the sodium or base balance which results in a lowered amount of sodium in the plasma. Recovery from "water intoxication" convulsions in animals is very rapid by the use of intravenously or intraperitoneally administered hypertonic sodium chloride solution. The observations in this paper show a complete contrast in the action of hypertonic saline on eclampsia compared with "water intoxication" in animals. Moreover, Stander, Eastman, Harrison, and Caddan²¹ have shown an increased amount of base in the plasma of eclamptics. This again is in contrast with the findings in "water intoxication" in animals. It would appear that, despite a similarity of the convulsive states in the two conditions, and that changes in the water balance are probably involved in both, a clear distinction must be made. McQuarrie,²² in his recent studies on epilepsy, has drawn attention to its similarity with eclampsia, as well as "water intoxication." According to the author the use of urea among other measures acts as a preventive against the seizures of epilepsy. Harding and Harris²³ have recently in-

vestigated the influence of urea solutions in "water intoxication" with negative results, and are thus inclined to dissociate it from epilepsy. Whether urea will be of value in the treatment of eclampsia and pre-eclampsia remains to be seen.

REFERENCES.

- ¹Harding, V. J., and Van Wyck, H. B.: *Journ. Obstet. and Gynaecol. of the British Empire*, 1926, 33, p. 17.
- ²Stander, J. H.: *The Toxemias of Pregnancy*, Williams and Wilkins, Baltimore, 1929, p. 61.
- ³De Wesselow, O. L. V.: *Journ. Obstet. and Gynaecol. of the British Empire*, 1922, 29, p. 21.
- ⁴Lazard, E. M.: *Amer. Journ. Obstet. and Gynecol.*, 1925, 9, p. 178; 1926, 12; *Amer. Journ. of Surg.*, 1927, 3, p. 433.
- ⁵Titus, P., Dodds, P., and Willets, E.: *Amer. Journ. Obstet. and Gynecol.*, 1927, 14, p. 89; 1928, 15, p. 303.
- ⁶Mussey, R. D.: *Ibid.*, 1926, 11.
- ⁷Eckelt, K.: *Zeit. f. Geburt. f. Gynäk.*, 1919, 81.
- ⁸De Wesselow, O. L. V., and Wyatt, J. M.: *Modern Views on the Toxemias of Pregnancy*, Constable and Co., 1924, p. 70.
- ⁹Bland, P. B., and Bernstein, M.: *Amer. Journ. Med. Sci.*, 1927, 173, p. 844.
- ¹⁰Stander, H. J.: *The Toxemias of Pregnancy*, p. 53.
- ¹¹Allen, F. M.: *Journ. Amer. Med. Assoc.*, 1920, 74, p. 1652.
- ¹²O'Hare, J. P., and Walker, W. G.: *Arch. Int. Med.*, 1923, 32, p. 283.
- ¹³Allen, F. M., and Sherril, J. W.: *Journ. Med. Research*, 1922, 2, p. 429; *Zeit. f. Geburt. u. Gynäk.*, 1916, 88, p. 325; *Zentralbl. f. Gynäk.*, 1921, 45, p. 129.
- ¹⁴Zangemeister, W.: *Zentralbl. f. Gynäk.*, 1925, 49, p. 225; *Lehrbuch für Geburtshilfe*, Leipzig, 1927; *Deut. med. Woch.*, 1921, 47, p. 549.
- ¹⁵Ginglinger, M. A.: *Bull. Soc. d'Obstet. et de Gynecol.*, 1925, 17, p. 100.
- ¹⁶Holtermann, C.: *Zentralbl. f. Gynäk.*, 1928, 52, p. 1916; *Arch. f. Gynäk.*, 1928, 133, p. 121.
- ¹⁷Siderow, W.: *Zentralbl. f. Gynäk.*, 1929, 52, p. 1006.
- ¹⁸Bell, J. W.: *Amer. Journ. Obstet. and Gynecol.*, 1926, 12, p. 792.
- ¹⁹Corwin, J., and Herrick, W. W.: *Journ. Amer. Med. Assoc.*, 1927, 88, p. 457; Corwin, J., Herrick, W. W., Valentine, M., and Wilson, J. M.: *Amer. Journ. Obstet. and Gynecol.*, 1927, 13, p. 617.
- ²⁰Weir, J. F., Larson, E., and Rowntree, L. G.: *Arch. Int. Med.*, 1922, 29, p. 306; Rowntree, L. G.: *Ibid.*, 1923, 32, p. 157; Green, C. H., and Rowntree, L. G.: *Amer. Journ. of Physiol.*, 1927, 80, p. 209.
- ²¹Stander, H. J., Eastman, N. J., Harrison, jun., E. P. H., and Caddan, J. F.: *Journ. Biol. Chem.*, 1929, 85, p. 233.
- ²²McQuarrie, I.: *Amer. Journ. Dis. Child.*, 1929, 38, p. 45.
- ²³Harding, V. J., and Harris, L. J.: *Proc. Roy. Soc. Can.*, 1930, 24 (in press).

FRACTURES OF THE PATELLA.*

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MANY years ago a London woman sustained a fracture of the patella; John Hunter records how, after months of treatment, the knee-joint presented the dual problem of extension limited by powerlessness, and flexion limited by stiffness. Two hundred years have elapsed, but we are still faced with Hunter's problem; the keynote of the treatment of injury of the extensor apparatus of the knee, whether it be an avulsion of the quadriceps, fracture of the patella, rupture of the ligamentum patellae, or separation of the tibial tubercle, is the restoration of extensor power without impairment of flexion movement.

Although fracture of the patella was one of the first bone injuries to come within the scope of operative treatment there are still many differences of opinion. Is operation essential? Should the bone be drilled and sutured, or is it sufficient to suture the quadriceps expansion? Which is the best method of bone suture, and the best suture material? Is splintage also necessary? When should movements be commenced? Is manipulation necessary?

Hitherto these questions have been considered purely from the point of view of bony union, but if our objective is not simply union of the fracture, but restoration of the joint to normal, and we also consider the recovery of flexion movement, different indications arise. It may be true that immobilization of a normal knee does not cause permanent stiffness, but if a knee-joint is injured, and particularly if an arthritic knee-joint is injured, the inevitable penalty of prolonged immobilization in extension is some degree of permanent limitation of flexion. It follows that fractures of the patella demand sufficiently

firm operative fixation to render external fixation unnecessary; this is particularly true of the elderly patient. Early movement is essential, and a bandage applied firmly enough to prevent haemarthrosis gives all the post-operative fixation required. The actual date when movements are instituted must be governed by the success of the operative fixation. It is my own practice to test this by flexing the knee before stitching the skin. If the fragments are firmly held, assisted movement is commenced the following day. If the fracture is comminuted and fixation imperfect, movement is deferred until the fourth week, and is preceded by gentle flexion of the joint under anaesthesia deep enough to ensure perfect relaxation of the quadriceps. With a fully relaxed quadriceps manipulation is quite safe during the first four weeks. Within that time the adhesions are still light, they offer no resistance, and give way by the mere action of gravity unaided by any pressure on the part of the operator. On the other hand, I regard manipulation at any time between the fourth and twelfth weeks as very unsafe. The fragments are not yet firmly united, and the adhesions are now so firm and resistant that even the strongest suture material is liable to stretch or snap and allow separation of the fragments. Manipulation which is deferred longer than four weeks should be postponed until there is clear evidence of bony union of the fracture.

Operative Methods.

If the necessity for early movement is accepted it becomes obvious that operation is essential, that the bone must be sutured as well as the lateral expansion, and that the suture material must be strong. Catgut and kangaroo tendon are not sufficiently strong, and silver wire is notoriously unsafe and liable to snap (Fig. 1). Piano wire, on the other hand, is much less fragile, and its flexibility has the further advantage of allowing a knot to be tied. My own preference is for an absorbable material, and thick, braided silk has many advantages. It is extraordinarily strong, will withstand a much greater strain than the contraction of the quadriceps, and is sufficiently elastic not to snap; it can be secured in any length, is easy to work with, and can be tied in a knot which, owing to the roughness of the braided surface, will not slip. Lange has shown that

* A paper read in opening a discussion on fractures involving the knee-joint, in the Section of Orthopaedics at the Annual Meeting of the British Medical Association, Winnipeg, 1930.