# FURTHER OBSERVATIONS ON PARENTERAL MAGNESIUM SULPHATE THERAPY IN CORONARY HEART DISEASE: A CLINICAL APPRAISAL

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Over 2 years have elapsed since the publication of a preliminary report on the value of parenteral administration of a 50% aqueous solution of magnesium sulphate in coronary heart disease, both in its acute and chronic stages.[1] On the basis of my experience with this form of therapy spread over a period of 23 years, during which time I have treated well over 200 cases, it was claimed that a considerable number of sufferers from angina pectoris were relieved of their symptoms by this form of therapy, the relief lasting up to 6 months after completion of treatment. It was also claimed that injections of magnesium sulphate were of inestimable value during an attack of myocardial infarction, greatly reducing the immediate mortality from the attack. Since the publication of this communication a number of reports have appeared in support of this form of therapy. Papenfus,[2] Agranat,[3] Teeger,[4] Marais,[5] Feldman,[6] Butler,[7] Shapiro[8] and Finn[9] all testified to the value of the treatment. Because of these encouraging reports it was felt that the time was ripe to describe my method of treatment of coronary heart disease with injections of magnesium sulphate in greater detail than before and thus enable those who would like to try this still unorthodox form of therapy to avoid mistakes and pitfalls which could only bring an otherwise valuable drug into disrepute.

Two distinct groups of patients will be discussed separately, viz. (*a*) patients suffering from chronic coronary heart disease and angina of effort, with or without a previous history of acute coronary episodes, and (*b*) patients who were first seen during an acute attack of coronary thrombosis or acute coronary insufficiency.

### GROUP A. CHRONIC CORONARY HEART DISEASE WITH ANGINA OF EFFORT

It is generally conceded that the evaluation of what is claimed to be a clinically effective therapeutic agent for angina pectoris is beset with extreme difficulty. Nevertheless, after 2 further years of observation by myself and others, it can now be stated with certainty that amongst patients with established coronary heart disease and angina of effort an appreciable number respond to parenteral administration of magnesium sulphate, sometimes in a dramatic and almost unbelievable manner, and this after all conventional and accepted methods of therapy had failed and sufferers had lost hope of ever obtaining relief. Two particularly gratifying case histories published to date are those of medical practitioners (neither under my care), both of whom were incapacitated by severe angina of effort; both had unsuccessfully tried every known form of therapy; both lost their symptoms and returned to normal life after having resorted to injections of magnesium sulphate.[2,6]

The number of patients who respond seems to vary. In a recent series of 27 of my cases suffering from angina 15 declared themselves satisfied with the results and eager or willing to carry on. In Agranat's series[3] of 50 patients treated 25 reported considerable improvement lasting from 1 to 9 months. Teeger[4] reported 13 improved out of 15 cases treated, Marais[5] 5 improved out of

6 treated, Butler[7] 25 improved out of 27 treated. Thus out of a total of 125 cases treated by 5 different practitioners 83 (or 66%) improved.

Russian workers have independently tried magnesium sulphate therapy in angina pectoris and reported gratifying results. Aphanasieva and Kirilova[10,11] were both favourably impressed by the response obtained in hypertensive patients, while Perlia[12] treated 79 cases suffering from severe angina with 77 (or 97%) improved.

*Rationale*

Since the publication of our report significant experimental work on animals which throws light upon the rationale of the treatment has been published by various workers. Bersohn[13] has recently reviewed fully the experimental work done. Still more recently, Selye[14] found that magnesium salts proved 'eminently effective' in the prophylaxis of artificially produced infarct-like lesions in the hearts of rats. These publications suggest that 'magnesium deficiency (probably conditioned rather than primary) may perhaps be more intimately involved in the whole picture of athero- and thrombogenesis than is usually appreciated'.[13]

In September 1956 Malkiel-Shapiro, Bersohn and Terner showed that abnormal lipoprotein patterns may rapidly revert to normal with parenteral administration of magnesium sulphate.[1] Butler[7] and Parsons[15] have recently confirmed this observation.

Despite these encouraging experimental and laboratory findings the rationale of the treatment still remains fairly obscure and many clinical facts require further elucidation. Thus, in a series of 22 of my cases suffering from angina pectoris, 14 showed a dramatic improvement in their lipogram simultaneously with their clinical improvement, 4 showed no improvement in their lipoprotein pattern but nevertheless improved clinically, while 4 did not improve at all.[1] It follows that no total parallelism could be observed between clinical improvement and the reversal of a disturbed lipogram to normal; nor is it clear why the response to treatment is not uniform, or why in carefully selected, seemingly identical, cases some derive the greatest benefit from treatment, while others do not respond. Thus it is fair to say that at the present state of our knowledge experimental and laboratory data are insufficient to guide the clinician in the selection of suitable cases, and that clinical experience and wise judgment have still to be relied upon to bridge the gap and enable us to make a good guess.

*Selection of Cases*

The following observations may be helpful in selecting cases suitable for parenteral magnesium-sulphate therapy.

1. Symptoms resembling angina may result from conditions other than coronary heart disease. Hence, in selecting cases in my private practice, every patient was first subjected to a careful examination and every effort was made to single out sufferers whose cardiac condition was complicated by gall-bladder disease, peptic ulcer, diaphragmatic hernia, blood dyscrasias, arthritis, etc. This was done with the aid of all the modern diagnostic methods at my command in Johannesburg. When the above-mentioned conditions were a contributory or complicating factor of established coronary heart disease, therapy was first directed towards the alleviation or if possible eradication of these ailments. Occasionally this was followed by marked amelioration or total disappearance of angina, rendering magnesium sulphate therapy unnecessary. However, the presence of gall-stones, peptic ulcer, hiatus hernia, benign anaemias or arthritis does *not* in itself constitute a contra-indication to parenteral magnesium sulphate.

2. The hypertensive, overworked, tense, worried and harassed individual, carrying a heavy load of responsibility, appears to respond better than the normo- or hypotensive, care-free and idle patient.

3. The individual whose daily calorific intake and sexual activities greatly exceed his physiological requirements responds better than the underweight, frugal and abstemious type.

4. The heavy smoker responds better than the non-smoker.

5. Patients who regularly consume large amounts of alcohol do particularly well on parenteral magnesium sulphate. The reason for this is not apparent.

6. Diabetics do well on magnesium-sulphate therapy.

7. Patients receiving long-term prophylactic therapy with heparin or oral anticoagulants do well on parenteral magnesium sulphate. A combined heparin and magnesium-sulphate therapy is particularly effective; these two drugs seem to have a synergic action.[1]

8. Patients over 50 respond better than younger patients.

9. Hyperthyroid cases do better on antithyroid therapy (i.e. the thio-uracil group of drugs or $^{131}I$).

10. Myxoedema, especially myxoedema induced as a therapeutic procedure, constitutes an absolute contra-indication to parenteral magnesium sulphate. In my experience the anginal syndrome of such patients may become greatly aggravated by this form of therapy.

11. Any form of malignancy constitutes a contra-indication.

12. Sufferers from essential hyperlipaemia respond poorly to magnesium sulphate.

13. Hyperparathyroidism seems to call for great caution and careful dosage if magnesium sulphate is to be given. I have given it to only one patient suffering from this complaint. A woman aged 40 had been suffering from hyperparathyroidism and was clinically cured after the removal of a parathyroid adenoma. Her serum calcium, however, remained fairly high. Three years later she sustained an attack of acute coronary insufficiency, for which she was given magnesium sulphate injections. After the third injection she developed a violent reaction (malaise, headache, pyrexia, arthralgia and neuritis) which lasted for 2 weeks. Her angina, however, greatly improved and it is possible that the dose of magnesium sulphate given was too high.

*Treatment of Chronic Angina Pectoris*

Magnesium sulphate may be administered either intravenously or intramuscularly.

Kutham,[23] who apparently was the first to publish his observations on parenteral magnesium sulphate in angina pectoris, favoured intravenous injections. I have been using the intramuscular route exclusively. This method, though apparently less effective, is less hazardous. It has the additional advantage of preserving veins of the antecubital fossa, which certainly would get thrombosed in the course of long-term magnesium-sulphate therapy.

Injections are given into the upper and outer quadrant of the buttock. Most patients experience a varying degree of local discomfort from the injection. It is usually similar in all respects to discomfort from any injection, although slightly more severe. It rarely lasts more than 30 minutes. The discomfort can be minimized by appropriate technique, which may be summarized as follows:

(*a*) Use a needle at least 1½ inches long.

(*b*) Use the empty-needle method.

(*c*) Syringes and needles should be sterilized by autoclaving or boiling; chemicals (e.g. alcohol) should never be used, because they may cause magnesium sulphate to crystallize out and the piston to stick.

(*d*) Select your site of injection as high up and as laterally as possible, where the gluteus medius is not covered by the gluteus maximus; thus you will prevent the fluid from entering the fascial sac which is covered by the gluteus maximus muscle and in which the sciatic nerve lies.

(*e*) Make a zigzag needle tract. (This is effected by insertion of the needle part way, with a change in direction and then continuation of the insertion of the needle to the hub.)

Treatment can be given intermittently or continuously over an indefinite period. If given intermittently, the injections are spaced at 5-day intervals and 12 injections constitute a course. Then treatment is interrupted and the patient instructed to return in approximately 4-6 months' time for a further course should he feel that he has benefited. If the injections are given continuously they are spaced at 7-day intervals. I had been using the intermittent treatment as a matter of routine. During the past 3½ years, however, a few advanced cases have been put on continuous therapy at their own request. One patient has had injections of magnesium sulphate weekly for the past 3 years. To date no ill effect has been observed.

The *dose in chronic angina* varies between 0·5 and 2 ml. of the 50% aqueous solution. With the continuous type of treatment I have never exceeded 0·5 ml. per injection. With the intermittent type of treatment the exact dose varies in each individual case, depending on sex, age, weight, height, and whether the patient is hypo-, normo- or hypertensive. The average single dose is 1 ml. Where the patient is hypotensive or in failure I have never exceeded 0·5 ml. per dose. It cannot be emphasized too strongly

that no useful purpose is served by giving injections more frequently or in larger doses. Two ml. should be given only to the hypertensive and obese patient. Overdosage will render the injection more painful and may cause depression and listlessness, which in turn will discourage the patient from persevering with treatment.

Russian workers favour the intravenous route. Kirilova[11] injected intravenously one ml. of a 10% solution of magnesium sulphate mixed with 9 ml. of a 40% solution of glucose. Perlia[12] gave 5 ml. of a 25% solution daily, and the number of injections varied from 1 to 12; thus up to 15 g. of magnesium-sulphate salt was infused intravenously in the course of 12 days. All Perlia's patients were hospitalized during treatment. Unfortunately, this author does not mention whether any adverse reactions were observed. This method of treatment may require hospital facilities and is probably not suited for the ambulatory patient. However, Perlia claims a very high percentage of response in the control of symptoms (97%), and I feel that his method merits careful consideration. It could be tried on patients who fail to respond to my low-dosage schedule by the intramuscular route.

*Patients' Response to Therapy*

Patients who respond to intramuscular injections of magnesium sulphate do so in a two-fold manner:

1. The frequency and severity of their anginal attacks gradually diminish, the tolerance to effort and to emotional stress increases, and some become asymtomatic for periods of many months, even after cessation of therapy.

2. Within 12 hours after the first injection some experience a feeling of profound well-being quite unrelated to their cardiac condition. This state of euphoria is most marked during the first course of treatment. It may recur to a lesser degree during the second course, but rarely thereafter. Patients who are given repeated courses over a period of many years may begin to feel listless and mentally depressed during the treatment, despite improvement of their anginal syndrome. Rightly or wrongly I have always interpreted this as a warning sign that 'saturation point' has been reached, calling for a drastic reduction in dosage (e.g. 1 ml. of a 1% solution) or temporary interruption of treatment. However, a recent experience makes me wonder whether this caution was really justified.

K.T., aged 54, manager of a large engineering firm, experienced a severe attack of coronary thrombosis in August 1944. He was treated with parenteral magnesium sulphate and bed rest for 3 months and made a good recovery. He returned to work leading an active and strenuous life. The only residual symptom was mild angina brought about by emotional stress or undue exertion. During a subsequent period of 14 years he received courses of parenteral magnesium sulphate regularly every 6 months. These benefited his angina and gave him a feeling of energy and euphoria. On 5 April 1958 he returned for yet another course of injections. On this occasion, however, after the 3rd injection he experienced a feeling of profound mental depression and listlessness. His blood pressure remained moderately elevated (160/100 mm. Hg) and his general condition remained unchanged. Despite my advice to the contrary, he insisted on carrying on with the treatment. After the 10th injection the cloud spontaneously lifted and he volunteered the information that this course of treatment was the most beneficial he had ever had. It is noteworthy that to date I have not observed these bouts of depression in patients who received magnesium-sulphate injections continuously.

With the exception of the one case of hyperparathyroidism referred to above, it has been my experience that parenteral magnesium sulphate in the dosage recommended is completely non-toxic, and I have never had reason to regret having given it to sufferers from chronic coronary heart disease. In the course of 25 years 5 of my cases developed pyrexia up to 102 F with malaise and arthralgia after the first two or three injections, the reaction lasting 24 hours. No further complication developed and all 5 reported great amelioration of their anginal syndrome.

Every patient who benefits from parenteral magnesium sulphate should be warned against assuming that the improvement in his clinical condition heralds the return of youth. On experiencing the disappearance or alleviation of angina coupled with a feeling of energy and euphoria these patients may be tempted to indulge in activities far in excess of the functional capacity of their hearts, occasionally with disastrous consequences.

W.H., aged 77, still at work in a large departmental store in Johannesburg, suffered a myocardial infarction in 1951. He was treated with parenteral magnesium sulphate and made a good recovery. He remained with a mild residual angina of effort as well as mild attacks of nocturnal angina; these were relieved by nitroglycerin and aminophyllin. Between the years 1951 and 1957 he was given courses of parenteral magnesium sulphate at approximately 6 months' interval; these benefited him a great deal. In November 1957, he came for yet another course of magnesium-sulphate injections because 'he felt he needed them'. Clinical examination was non-contributory, blood pressure was 150/90 mm. Hg and his cardiogram showed no deterioration compared with previous tracings. He commenced a course of treatment with parenteral magnesium sulphate on 2 November 1957, and once again he responded in a dramatic manner. By 13 December he felt so well that he ventured to lift a heavy case of merchandise. The same night he sustained an anterior infarct. Parenteral magnesium sulphate was immediately supplemented with heparin (5,000 units *b.d.*). He remained in bed on a combined treatment of magnesium sulphate and heparin till the end of February 1958. By the end of March he had recovered sufficiently to return to sedentary work in his store.

Some medical men have questioned the efficacy of magnesium-sulphate therapy on the ground that it does not cure every patient, or that the relief obtained is only partial and relatively short-lived. This is by no means a valid objection. We might as well repudiate general medicine because it cannot convert every middle-aged invalid into an A1 physique. Any method of treatment which helps a patient to be moved from one stage of disability to a lesser one merits careful consideration. One does not talk of the 'cure' of a case of congestive heart failure by digitalis therapy, but nevertheless admits its value.

GROUP B. IMPENDING AND ACUTE MYOCARDIAL INFARCTION

I have been treating my patients suffering from acute myocardial infarction with injections of magnesium sulphate since November 1933. While it is not possible to give the exact number of patients treated by this method, at a most conservative estimate 50 *proved* cases under the age of 70 and many more suspected cases have been treated between 1933 and 1955. All my patients were seen *early*, usually within 3 hours of the onset of their symptoms. I believe that early treatment with magnesium sulphate is of utmost importance if uniform success is to be achieved, and I have rigidly adhered to the dictum, 'if you are in doubt about the diagnosis, play safe and treat the patient for acute coronary thrombosis, unless you have absolutely ruled out this possibility'. At no time did I allow positive clinical evidence to be nullified by negative electrocardiographic findings. I am convinced that in dealing with this treacherous condition the survival of the patient depends to a greater degree on the clinical judgment and skill of the general practitioner, who sees the patient early, than on all the experience and knowledge of the cardiologist, who is often called in hours or even days later, when the fate of the patient has virtually been sealed.

Magnesium-sulphate injections do not prolong clotting time, nor do they depress the prothrombin index.[1] Hence they cannot precipitate or aggravate an existing haemorrhage. Thus, should the diagnosis of coronary thrombosis have to be revised at a later date in favour of an abdominal emergency requiring surgical intervention, no harm has been done and operation can safely be performed.

Between the years 1933 and 1956 none of my patients under 70 treated with magnesium sulphate died in an attack of coronary thrombosis. During the past 24 months further 14 proved cases of myocardial infarction or acute coronary insufficiency were treated, with 1 death; 11 were men and 3 were women. The ages of the 13 who survived were: 47, 50, 51, 52, 52 (second attack), 55 (second attack), 57 (third attack), 58 (second attack), 58 (second attack), 60 (third attack), 69 (second attack), 77 (second attack) and 79.

The fatal case was a relapse. He was 59 at the time of death. He had experienced his first episode of myocardial infarction 2 years previously, when he was treated with parenteral magnesium sulphate and intravenous heparin; he tolerated both drugs well and made a good recovery. In this, his second

attack, treatment was withheld for approximately 6 hours after the onset of symptoms. When seen he was moribund. He died suddenly, presumably from cardiac arrest, 30 minutes after an intramuscular injection of 0·5 ml. of magnesium sulphate and 3 minutes after an intravenous injection of 5,000 units of heparin.

Thus, in the present series of at least 64 proved cases under the age of 70 treated with parenteral magnesium sulphate over a period of 25 years, the immediate mortality rate (i.e. within 4-6 weeks after onset) was only 1·6%, while the average mortality rate given by most modern workers for patients who were treated by conventional methods varies from 19% to 50%.[19,20]

*Treatment of Acute Myocardial Infarction*

Unless the patient is in severe cardiac shock the first injection of magnesium sulphate is given immediately the patient is seen. This first dose should never exceed 0·5 ml. of a 50% solution. The second dose is given 12-24 hours later. If the blood pressure is low this second dose should again not exceed 0·5 ml. If the blood pressure and pulse pressure are normal, 1 ml. is given on this second day of treatment. Thereafter 1 ml. should be administered on the 4th, 7th, 10th and 14th days, and then 1 ml. is injected every 5th day till the patient is discharged. Morphia or pethidine are given *only* if pain is severe and the conventional management of coronary thrombosis is strictly adhered to. In other words, in the acute phase of coronary heart disease parenteral magnesium sulphate should be given concurrently with and *not* as a substitute for any therapeutic measure which the physician may deem necessary to institute in order to save his patient's life.

If the patient is in severe cardiac shock, with its well-known clinical features of prostration, pallor, sweating, vomiting, coldness of extremities, a small thready pulse, and low blood pressure and low pulse pressure, treatment with magnesium sulphate (and heparin) should temporarily be postponed and attempts should first be made to raise the blood pressure by appropriate means.

The diminution of coronary blood flow which occurs in shock following myocardial infarction[16] greatly impairs the efficiency of the uninfarcted myocardium and aids in the establishment of a vicious circle whereby low blood pressure reduces coronary flow, which still further impairs myocardial contractility, decreasing the stroke volume and the output of the heart.[17,18] Under this desperate condition, which is reputed to carry an 80% mortality rate,[20] it may be wiser to start therapy by first administering mephentermine or L-noradrenaline in order to restore the cardiac output, and to administer magnesium sulphate and heparin only after sustained improvement in the circulation has been achieved.

Adhering strictly to the above rules of treatment, it has been my experience that magnesium sulphate is a safe drug to use in acute myocardial infarction. It has been said that the magnesium ion is toxic when high concentrations accumulate in the extracellular fluid[21] and I have always been careful to employ a low dosage schedule.

Agranat,[3] in a recent review of 50 cases of coronary heart disease which he treated with parenteral magnesium sulphate (2 ml. per dose), decided against its use in acute coronary thrombosis because of its possible depressant effect on respiration and blood pressure when given in large doses. He quotes 3 of his cases who died during the early stages of magnesium-sulphate therapy, one of the three having had pethidine and heparin administered at the same time. It is not clear why these sudden deaths, which might have occurred in any event, should have been attributed to magnesium-sulphate injections. I feel that his unfortunate experience, should it really be attributable to parenteral magnesium sulphate, calls rather for a reduction of dosage than total abandonment of so valuable a drug. Most, if not all, therapeutically effective drugs are potentially hazardous. Deaths have followed the administration of digitalis, quinidine, aspirin, penicillin and anticoagulants, to mention only a few, yet these drugs are in common use because their therapeutic effectiveness far outweighs the risk involved.

## CONCLUSIONS

To be classed as therapeutically effective in the field of coronary heart disease a drug must fulfil at least one of the following criteria: It must be effective in relieving symptoms of angina pectoris; it must reduce the mortality rate in acute myocardial infarction; it must prolong the life expectancy of those who have recovered from an attack of acute coronary thrombosis. What then, in summary, can parenteral magnesium sulphate, in the light of our present knowledge, do for the sufferer from acute and chronic heart disease?

1. It alleviates to a varying degree the symptoms of angina pectoris in at least 60% of carefully selected cases. This now appears to be certain.

2. It ranks supreme in the treatment of impending myocardial infarction.

3. In my experience it has greatly reduced the immediate mortality of acute coronary thrombosis in patients who have not outlived their normal span of life. Hitherto acute myocardial infarction carried an immediate mortality of 19-50%. In my series of at least 64 cases only one died. I hold that by early recognition of impending coronary thrombosis and its prompt treatment with parenteral magnesium sulphate a large number of patients can be saved. This statement awaits corroborative evidence from others, as the number of my cases is small. Once again, the importance of early treatment cannot be over-emphasized. There is nothing unusual about this observation. It applies with equal force in many other clinical emergencies and to many other drugs. It is the cry of all medical practitioners that if only illness could be 'got at' at an early stage therapeutic prospects would be so much improved.

4. Finally, does long-term magnesium-sulphate therapy prolong the life expectancy of those who have recovered from an attack of coronary thrombosis? Clearly I cannot attempt to answer this question on the basis of my limited experience. My opportunities of studying the long-term effect of parenteral magnesium sulphate have been only those which a private practice mostly amongst the well-to-do afforded. While controlled long-term therapy is the only way to assess the value of parenteral magnesium sulphate in increasing the life span of sufferers from chronic coronary heart disease, it is clearly quite unrealistic to think of a private practitioner treating his coronary patients solely by long-term magnesium-sulphate therapy. Long-term therapy must have the status of a research weapon primarily. Patients who can be treated in this way would provide a rich reward of understanding on which sound preventive medicine could be based. Thus, the answer to this last question should be left to those who work in large teaching hospitals. They have a vast clinical material at their disposal and in consequence could carry out a scientifically controlled long-term experiment.

In designing such experiments the investigators should, in my opinion, beware of administering 'inert' injections and using the 'double blind' technique. No injection can be classed as a truly inert placebo, for it may release a chain reaction in the body, the nature of which will be totally unpredictable and unknown to the investigator. One of the least suitable placebos to use in these cases is saline. For many years past hypertonic saline has been used with good results in cases of angina not improved by rest and conventional therapy.[22] The assessment of survival by 'patient-years' amongst the magnesium-sulphate-treated sufferers, matched against a comparable group who receive no treatment, constitutes to my mind the only fruitful and constructive line of investigation. This method has been regularly used by all workers attempting to assess the value of long-term anticoagulant therapy in prolonging the life span of coronary patients, and it is hoped that controlled investigations on these lines with parenteral magnesium sulphate will soon be started in our large teaching hospitals.

No claim is made that parenteral magnesium sulphate will resuscitate the dying, nor that it is a complete treatment of coronary heart disease. It is not intended that it should supersede older and well-tried remedies. Whenever possible, I have given it concurrently with other drugs. Thus, I have found the concurrent administration of heparin and magnesium sulphate to be particularly helpful. These two drugs have a synergic action[1] and during the past 3 years I have been using, whenever possible, magnesium sulphate and heparin concurrently in an effort to prolong the life of those who have recovered from one or more attacks of coronary thrombosis. I have trained the intelligent and cooperative type of patient (or his wife) to inject heparin in the same way as a diabetic is trained to inject insulin. When given concurrently with magnesium sulphate the dose of heparin required is small and the injection safe and relatively painless. The patient is instructed to give himself 5,000 units of heparin once a day whilst the doctor (or the nurse) administers from

0·5 to 1 ml. of magnesium sulphate intramuscularly once a week. After 3 years of experience with this form of therapy it is my belief that, at the present state of our knowledge, the skilful concurrent administration of these two drugs constitutes the method of choice in the prophylaxis and treatment of coronary heart disease both in its acute and chronic stages. I have little doubt that it is superior to long-term therapy with oral anticoagulants. Moreover, it can be carried out in the remotest village without the aid of laboratory investigations.

As this paper has been written mainly for the benefit of the clinicians, I have omitted any discussion of the many animal experiments performed and theories recently advanced in support of magnesium therapy in arteriosclerosis. By the same token I have given a complete account of various techniques in the hope that interested readers will begin the clinical study of parenteral magnesium-sulphate therapy. There is still much to be learned about this our new asset in the treatment of coronary heart disease.

SUMMARY

1. The value of parenteral magnesium-sulphate therapy in acute and chronic coronary heart disease has once again been affirmed.

2. 125 cases of angina have been treated by 5 workers with 66% remission of pain.

3. 64 cases of acute coronary thrombosis or acute coronary insufficiency have been treated. Of these only one died in an acute attack. The great importance of *early* parenteral magnesium-sulphate therapy in these acute cases has been stressed.

4. It is suggested that in cases who have recovered from an attack of coronary thrombosis, life expectancy can be improved by combined heparin and magnesium-sulphate long-term therapy.

5. Details are given of techniques, indications, contra-indications, dosage and toxicity.

6. A plea is made for a scientifically controlled investigation on a large scale in teaching hospitals.

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