DICOUMAROL IN ACUTE CORONARY OCCLUSION*

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A STUDY of all cases of acute coronary thrombosis admitted to the medical wards of the Montreal General Hospital between January 1946 and May 1948 has just been concluded. One of the main purposes of this study was to investigate the effect of dicoumarol on the overall mortality and on the thrombo-embolic phenomena associated with this disease. During the first sixteen months of this period, alternate cases were given dicoumarol; the remainder, receiving only supportive therapy, served as controls. During the following thirteen months, all cases were given dicoumarol, as the increasingly favourable reports in the literature on this form of treatment appeared to render the original course unjustifiable.[1 to 4]

In this manner, a total of 57 cases of acute coronary occlusion with myocardial infarction were studied, 38 being given dicoumarol, and 19 receiving only supportive therapy. In order to increase the control series to a number equal to that of the treated group, an additional 19 cases were picked at random from the 1945 records of this hospital. This brought the total number of cases to 76, half of which received dicoumarol and all of which were given the same supportive therapy.

All cases which died within 48 hours of their initial attack were excluded from both the treated and the control groups, as dicoumarol could not be expected to influence the course of the disease during this period.

Methods employed.—In those cases treated with dicoumarol, the dosage used was that described by Barker and his associates.[5] A 300 mgm. dose was given orally as soon as the presumptive diagnosis was made. Single doses of 100 to 200 mgm. were then given whenever the prothrombin time was below 35 seconds.

Prothrombin time determinations were carried out using Quick's method.[6] Thromboplastin was prepared weekly from the brain of a freshly killed rabbit. With this thromboplastin, normal values for prothrombin time were 15 to 22 seconds.

Results.—Of the total of 76 cases investigated 19 showed thrombo-embolic complications, including extensions or recurrences of their original coronary occlusion. These are shown in detail in Table I. It will be seen that in the control series, 12 cases (31.6%) had complications, while in the treated group only 7 cases (18.4%) showed such occurrences. Two cases showed more than one thrombo-embolic phenomenon.

TABLE I.

TOTAL COMPLICATIONS—FATAL AND NON-FATAL

	Controls		*Treated*	
Complications	*Fatal*	*Non-fatal*	*Fatal*	*Non-fatal*
Pulmonary infarction	2	1	1	2
Mesenteric infarction	–	–	1	–
Renal infarction	1	–	–	–
Thrombosis of leg veins	–	1	–	–
Arterial embolus (1 radial)	–	1	–	–
Cerebral embolus	–	–	–	1
Secondary coronary occlusion or extension	4	2	2	–
Total thrombo-embolism	7	5	4	3
Other complications				
Congestive heart failure	–	–	1	–
Sudden death (post mortem showing only the coronary occlusion)	2	–	–	–

A review of the 7 treated cases which developed thrombo-embolic complications or extensions shows that with one exception, all had lapses in their prothrombin times to sub-therapeutic levels within 48 hours prior to the complicating episode. The remaining case was well controlled throughout the course of treatment, prothrombin time never falling below 36 seconds after the 5th day in hospital.

Fatal thrombo-embolic complications or extensions occurred in 7 (18.4%) of the control series, and in 4 (10.5%) of the treated cases. In addition to these, two of the control series died suddenly, but at autopsy revealed no lesion other than the original myocardial infarction, and one of the treated cases died of congestive cardiac failure. The overall mortality was therefore 23.7% in the control series, and 13.2% in the treated cases.

SUMMARY AND CONCLUSIONS

1. A series of 76 cases of acute coronary thrombosis with myocardial infarction has

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been studied, half of the cases being treated with dicoumarol.

2. The incidence of thrombo-embolic phenomena (including extensions of the original lesion) was 18.4% in the treated cases as compared with 31.6% in the control series.

3. Of the 7 treated cases which developed thrombo-embolic phenomena, all but one had lapses in their prothrombin times to sub-therapeutic levels just before the occurrence of the complications.

4. The overall mortality rate in our series was 13.2% in the treated cases as compared with 23.7% in the control group. The mortality rate due directly to thrombo-embolic phenomena was 10.5% in the treated group, and 18.4% of the control series.

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RÉSUMÉ

Etude d'une série de 76 cas d'infarctus du myocarde, dont la moitié ont été traités par le dicoumarol. Celui-ci a été donné en doses de 100 de 200 milligrammes, de manière à maintenir de temps de prothrombine au-delà de 35 secondes. Dans la présente série, les complications de l'infarctus du myocarde par occlusion coronarienne ont été des embolies pulmonaires, mésentériques, rénales, cérébrales, radiales; la thrombose des veines des membres inférieurs; des occlusions coronariennes secondaires; l'insuffisance cardiaque (un seul cas) et la mort sans autre complication. On le voit, le plus grand nombre de ces complications reconnaissent pour cause la mobilisation du caillot ou son extension. On s'explique ainsi que leur fréquence ait été plus grande dans les cas témoins (31.6%) que chez les malades ayant reçu le dicoumarol (18.4%). Chez ceux-ci, la mortalité est également moindre (13.2% au lieu de 23.7%). Il est intéressant d'observer que les accidents énumérés ci-haut sont presque toujours précédés d'un retour du temps de prothrombine à un niveau sub-thérapeutique.

PAUL DE BELLEFEUILLE

A contributor to the Vienna *Klinische Wochenschrift* notes that flies have been eradicated in the canton of Wallis and the city of Lucerne in Switzerland by DDT spraying for the last two years, with no effect on the prevalence of infantile paralysis. Most authorities consider flies of no significance in the spread of this disease but obviously there are plenty of other reasons for fly eradication.—*Hygeia*, **27**: 74, 1949.

REFLEX SYMPATHETIC DYSTROPHY

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FOLLOWING various kinds of trauma, and in some inflammatory states, there is sometimes observed an abnormal vascular reaction, intimately associated with pain. This reaction, originally physiological in character, becomes pathological when the phenomena are exaggerated in degree or prolonged in duration.

This local vascular response is activated through the agency of the autonomic nerves. It results in a disturbed nutrition. That it is reflex in character is now well established by both experimental and therapeutic experience. Therefore the term "reflex sympathetic dystrophy", used by many of the American workers, may aptly describe the various conditions, some of the more familiar of which are listed below.

Traumatic œdema or vasospasm.
Sudeck's atrophy, or post-traumatic osteoporosis.
Causalgia of the true classical type, described by Weir-Mitchell.
Minor causalgia, described by Homans.
Post-traumatic pain syndromes.
Thrombophlebitis and post-thrombophlebitic œdema.

Tender scars or other "trigger areas".
Muscle spasm, cramps, weakness.
Coldness and sensitivity to cold environment.
Discoloration of skin, mostly cyanotic.
Excessive sweating, trophic changes.
Hyperalgesia and hyperæsthesia, elevated skin temperature.
Pain, constant, spreading, with exacerbations, not corresponding to segmental nerve distribution.
Swelling and œdema.
Osteoporosis and joint stiffness, ankylosis.
Mental anxiety, pain related to emotional state.

Dr. Takats[1] assures us that all of these varying states are essentially similar in nature, but that they represent different manifestations of the one fundamental pathological process. So that of the symptoms and signs common to the group as a whole, one or more will dominate the picture according to the kind of injury, its duration and location, and the personality type of the patient.

A brief account of a few of these conditions, in patients who have recently come under our care, will serve to demonstrate some of the important principles pertaining to the recognition and treatment of this syndrome.

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