stitutional disturbances are wrought by this "break" referred to. But worst of all are the heart complications which every physician looks for, as a rule, in these affections. What I have stated with regard to the possible etiology of the inflammation of joint-structures applies equally to the membranes of the heart. I do not deem it compatible with the plan of this effort to enter into the subject of *complications*, other than merely to mention the fact that pericarditis, endocarditis with consequent valvular disease, myocarditis and the formation of fibrinous deposits in the cavities of the heart, pleurisy, pneumonia, or bronchitis, are all liable to occur as complications of this disease. To these may be added peritonitis, which is rare; rarer still are cerebral and spinal meningitis. I may mention here before I forget, that the fact that lactic acid has been injected into the peritoneum and other serous cavities, producing the genuine rheumatic symptoms and pathological sequelae, only corroborates, as it seems to me, the view taken as to the ultimate pathological cause of rheumatic fever. In this case the lactic acid having been introduced stealthily, so to speak, and not in the natural way, it never has been, nor, as I believe, can it ever be a factor in this vitally chemical process of calorification, and hence is an intruder which must be got rid of. Hence too, (I shall have to speak more at length on this score when I come to treatment,) the injustice done to the venerable "vis medicatrix naturæ" to assert that the alkaline treatment is what must be looked to in most cases to effect a cure. That it has its place, and deservedly so, I deny not. But it is only a *chemical* remedy as it regards the specific poison in this affection. But more of this presently. As to the hyperinotic state of the blood in rheumatic fever, it should be noted this does not necessarily mean a superabundance of fibrin in the system. If it were so, an occasional sequela of rheumatic fever, and one which came under my notice at the Montreal General Hospital, would not be likely to occur—I speak of *purpuric extravasation*.

In illustration, the following case might be related: A young man, about 22 years of age, well nourished, &c., was admitted into the hospital in January, 1874, and was found to be suffering from acute rheumatism. Many of the symptoms above mentioned were well-marked in his case. Besides, there was developed about the third day after admission a distinct systolic endocardial murmur, whiah was soon determined to be *mitral regurgitant* (which persisted for some time after he left the hospital, as I had the opportunity of ascertaining for myself). One morning during his convalescence, he happened to stand for a few minutes beside his bed. In a short time after, the lower part of his left leg presented a well-marked *purpuric extravasation*. The attending physician told us it was the result of his weakened condition. No doubt it was; but that young man, even after he got strong, continued to manifest the *hemorrhagic* tendency—bleeding at his gums was a common occurrence, as he assured me more than once. It is beyond my province here to enter into an investigation of the etiology of purpura. But I may state that his blood was evidently *aplastic*, and hence, although there might have been an hyperinotic condition of his blood, the supposition that this was due to its not being deposited in the tissues on account of the systemic derangement is alike possible. But the more probable view as to it, is this:—From the deranged calorific process as above described taking place, animal heat must be supplied from some source, and hence the proteinaceous compounds, the fibrin-forming elements, are called into requisition to supply it. I think there can be little doubt that the formative process at this time is much diminished if not almost checked; hence the materials that are normally used in this process are by a retrograde metamorphosis called into requisition to supply heat. And the oxidation of these tissues always result in abnormal increased temperature (fever). And hence, perhaps, the hyperpyrexia so common in this disease. It may be noted too, here, that the purpura hemorrhagica, which is sometimes seen during convalescence, &c., is probably due to a weakened state of the capillaries as a result of this interference in the *formative process*, their minute vessels rupturing on the patient's getting into the erect posture for instance, and causing the sub-cutaneous extravasation. The diminution in the number of the red blood corpuscles has also been observed, which may be due to this formative interference. In fact it is in this disease that is to be particularly observed—a process of "back-working"—the reversal of the engines in a measure. The subacute variety of this affection now demands a little consideration.

This is often a very troublesome complaint There is very little pyrexia; but in some instance the acid sweats of the acute variety are present, though to a much less extent; one or more joints become affected for a long time, the conditions being almost stationary. Exacerbations are not uncommon, and they are liable to occur from slight causes or without any evident cause at all; but I would here state it as being very probable, as it seems to me, that the predisposing cause above mentioned in