Date: \_\_\_/\_\_\_/\_\_\_\_\_ Pt#: \_\_\_

Laboratory Results/Interview

Interviewer: RM BH LG SZ WA KS JB BB MM

(In person, by Phone, Office, Hospital, Nursing Home, With Relative)

(*Circle Appropriately*)

The majority of the testing of your blood was carried out on the DNA extracted from your white blood cells. Polymerase chain reactions (PCR) tests were carried out on genes, which regulate the coagulation proteins we wanted to study. These tests tell us whether or not you have inherited a double dose of a particular mutant gene from your mom and dad (*homozygous)*, or, a single dose from mom or dad (*heterozygous*). Thrombophilia is an increased tendency to form blood clots that can be inherited. Hypofibrinolysis can also be inherited. This is the reduced ability of the body to dissolve blood clots. One or both of these conditions could be contributing to your jaw problem. Identifying these specific markers might help explain the necrosis and pain you have experienced in your jaw. Treatment with blood thinners may prevent further deterioration. If blood thinners are indicated, they will only be prescribed by your Primary Care Physician or Oncologist.

GENETIC CAUSES of THROMBOPHILIA that were tested:

1. Innate resistance to activated protein C is a hereditary defect in the Factor V gene (Leiden mutation). Your *Factor V Leiden gene* is:

\_\_\_ Normal

\_\_\_ heterozygous (8X more likely to form abnormal blood clots) \_\_\_ homozygous (80X more likely to form abnormal blood clots)

1. Mutation in the Prothrombin gene (Factor II) can increase the risk of blood clots. Your *Prothrombin gene* was: \_\_\_ normal \_\_\_ heterozygous \_\_\_ homozygous
2. Mutations of the MTHFR are either homozygous or heterozygous. These mutations can be associated with elevated Homocysteine levels in the blood, which can lead to an increase in clotting tendency.

Your C677T *MTHFR gene* was: \_\_\_ normal \_\_\_ heterozygous \_\_\_ homozygous

Your *A1298C MTHFR gene* was: \_\_\_ normal \_\_\_ heterozygous \_\_\_ homozygous

*Attachment 8*

Pt#: \_\_\_

GENETIC CAUSES of HYPOFIBRINOLYSISthat were tested:

1. The major factor governing the ability to dissolve blood clots is Plasminogen Activator Inhibitor (PAI-1 Activity*).* The upper normal limit is 21.1 u/mL.

Your *PAI-1 Activity* level was: \_\_\_\_\_\_\_u/mL \_\_\_ normal \_\_\_ high \_\_\_ low

1. Often, high PAI-1 Activity is caused by an inherited defect in the PAI-1 gene. If you have this mutation or, if your PAI-1 Activity is high, then, once the body forms a blood clot, your body has trouble dissolving it.

Your *PAI-1 gene* was: \_\_\_ normal \_\_\_ heterozygous \_\_\_ homozygous

1. The third hypofibrinolytic factor, which we measured is Lipoprotein *(a).* If the level of Lipoprotein (a) is high, this makes it more difficult for the blood to dissolve blood clots. The upper normal level is less than 35 mg/dL.

Your Lipoprotein (a) level was: \_\_\_\_\_\_\_mg/dL \_\_\_ normal \_\_\_ high \_\_\_ low

NON-GENETIC CAUSES of HYPOFIBRINOLYSIS that were tested:

Homocysteine is an amino acid that can increase blood clotting in both arteries and veins. The normal level is < than 14.1, but we prefer the level to be 13.1 umol/L.

Your *Homocysteine* level was: \_\_\_\_\_\_\_\_umol/L \_\_\_\_\_\_normal \_\_\_\_ high

*Attachment 8*